

ANNALS  
OF  
OTOLOGY, RHINOLOGY  
AND  
LARYNGOLOGY

INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

---

---

VOL. XXXIII.

DECEMBER, 1924.

No. 4.

---

---

LXVIII.

FOREIGN BODIES IN THE AIR AND FOOD PAS-  
SAGES. CHARTED EXPERIENCE IN CASES  
FROM No. 631 TO No. 1155, AT THE  
BRONCHOSCOPIC CLINIC.\*

By CHEVALIER JACKSON, M. D., Sc. D.,  
PHILADELPHIA.

One of the greatest shortcomings of medical literature is its unavailability for instant reference in urgent, daily, clinical work. When a surgeon meets with disaster he has the literature searched and there is brought to him a stack of pamphlets and journals that require weeks for perusal. In them he finds that other surgeons have met the same disaster and have suggested means of avoidance. This is learned *after*, not *before* disaster, which has perhaps cost human life. The surgeon perhaps publishes his case with a review of the literature and another warning. This in turn is buried.

In this paper I shall endeavor to show how we have all of our past experience at instant command for review *before* we do an endoscopy on a similar case.

When a case comes to the Bronchoscopic Clinic we get out records of previous cases of the same kind for study. We note the difficulties encountered and how they were solved. We are thus forewarned and forearmed. We rehearse the technic used and in some cases improvements in technic are developed by study of the problem on the rubber tube, cadaver

---

\*Awarded the Research Fund Prize by the American Laryngological, Rhinological and Otological Society, 1923.

and dog, and in any case we have our previous observations by which to profit.

For instant availability we have tabulated the cases and this has usually obviated the necessity of going over cumbersome, voluminous records, which, however, are also available if needed for supplementary study. When a peanut case comes in it requires 20 minutes to review our difficulties in the preceding 80 cases of peanut kernels. We colloquialize the tables as "canned experience."

Thinking these tables might be equally useful to other endoscopists for ready reference, covering as they do practically every class of foreign body, they are here presented for publication. It is confidently expected that by serving as a starting point for other endoscopists there will be a continued progress in technic. If similarly arranged tables are made out by other endoscopists, we shall be glad to exchange with them in order to profit by their experiences as well as our own.

*System of Recording*—Because of the fundamental importance of establishing the status of the new field of work, endoscopy, it was deemed best to exclude all cases in which no endoscopy was done. Data on the excluded cases have been collected and will be published separately. When a foreign body case arrived at the Clinic it was not assigned a number until an endoscopic procedure of some kind was done. One instance of the usefulness of the here excluded nonendoscopic data may be cited. Of 18 cases of fishbones in the upper food passages, 14 were found in the crypts of the tonsils. Hence in any patient coming in complaining of feeling a fishbone in the throat, even though referred, as it often is, to a point low in the neck, the tonsils are searched very carefully.

The following tables include the endoscopic foreign body cases at the Bronchoscopic Clinic since the two previously published tabular reports,\* which gave the cases up to their respective dates.

In using these tables as a guide to work on similar cases the following points should be borne in mind:

1. The age of the patient is of the utmost importance in every phase of endoscopy, and should always be taken into consideration.

2. The duration of an endoscopy is recorded in our work in the operating room for two reasons. (a) Prolonged endo-

\*Peroral Endoscopy and Laryngeal Surgery. Text-book, 1914; and Mutter Lecture, Surgery, Gynecology and Obstetrics, March, 1919; also Proceedings College of Physicians of Philadelphia, 1917.



scopies in children under two years of age are dangerous. (b) Having a stop-watch running keeps us ever mindful, in preparing for, as well as during endoscopy, that time wasting must be eliminated. We want all the time available devoted to work with the eye at the tube. The inclusion of recorded time in these published tables, however, is chiefly to serve as an index of the difficulty we encountered and as a criterion of the efficiency or inefficiency of the particular method used in each instance.




3. The size of tube used will serve as a useful guide for selection of a suitable tube for similar cases, bearing in mind the age of the patient and the location of the foreign body in the tracheobronchial tree. The deeper the foreign body the smaller the diameter and the greater the length of the tube required to reach it.




4. The "Problem," "Forceps" and "Point of Seizure" taken together will serve as a forewarning of what may be expected in a similar case and will serve as a guide for practice on the rubber tube manikin before undertaking a similar case. The age of the patient as bearing on the size of the invaded bronchus is a fundamental consideration in simulating a problem with a rubber tube of corresponding size.

5. The pathology noted will serve as a forewarning of difficulties likely to be added by length of sojourn, though in many cases the pathology present was due to the instrumentation of our predecessors, in the respective cases. As we could not always distinguish with certainty the instrumental trauma of our predecessors from the trauma inflicted by the foreign body itself, we deemed it best to omit mention even of the obviously instrumental trauma inflicted by our predecessors. This trauma, along with the resultant pathology, enormously increased our difficulties and in some instances added to our mortality. In about 40 per cent of the cases here tabulated removal had been attempted before the patients were sent to us. All mention of this has been omitted. We have recorded in each case our own work only.



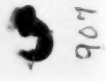
Note: The specimens of foreign bodies concerned in the cases, the data of which are tabulated in the following pages, were conveyed by deed of gift to the College of Physicians of Philadelphia. This institution has placed them in the Mutter Museum under the name of "The Chevalier Jackson Collection." To this has been added the subsequently removed foreign bodies up to No. Fbdy. 1402. The specimens and additional data are accessible to reputable members of the medical profession, for study, upon application to the Committee of the Mutter Museum, of which Dr. George H. Fetterolf is chairman.




## HARDWARE

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 668  668	22 mos.	Tack	Right inferior lobe bronchus, 87 days	None	5 mm.	Disengag- ing point	Side- curved	Near point inward rotation method	Extraction Cure	2 min. 30 sec.	Oral	Point of tack buried up to middle of flank in bron- chial wall. Localized inflammation. Intense sub- glottic edema.
Fbdy. 801  801	36 yrs.	Tack	Glossopiglotilean fossa	None					Extraction Cure		Oral	Tack with one quarter of shaft buried in left side of anterior surface of epiglottis.
Fbdy. 941  941	14 mos.	Tack	Right bronchus, 9 days	None	5 mm.	Point and shaft of tack in mediasti- num. Medias- tinal em- physema	Point turned out with side- curved for- ceps, re- moved with Tucker	Shaft after pushing head down	Extraction. Death from media- stinal emphy- sema	5 min. 56 sec.	Oral	Point of tack found buried in right internal wall.




	<p>12 yrs.</p>	<p>Two thumb tacks</p>	<p>Right main and stem bronchi respectively, 1 day</p>	<p>None</p>	<p>6 mm.</p>	<p>Two sharp pointed tacks senting tacks</p>	<p>Side-curved</p>	<p>Point of each tack separately</p>	<p>Extraction Cure</p>	<p>1st tack 45 sec. 2d tack 24 min. 3d tack 3 min. 43 sec.</p>	<p>Oral</p>	<p>First thumb tack encountered in right bronchus at orifice of upper lobe bronchus. Seized with side-curved forceps and removed. Reinsertion of bronchoscope located second tack with point directed at the entire bronchial lumen being shut off by the head of the tack. Turned edgewise with side-curved forceps and removed. Reaction confined to lower lobe of lung. Left bronchus appeared to be normal as its orifice was noticed in passing the bronchoscope downward into right bronchus.</p>
	<p>4 yrs.</p>	<p>Carpet tack</p>	<p>Lower lobe of right lung, 39 days</p>	<p>None</p>	<p>5 mm.</p>	<p>Fbly. buried in granulations</p>	<p>Side-curved</p>	<p>Shaft, inward rotation method</p>	<p>Extraction Cure</p>	<p>35 min. 10 sec.</p>	<p>Oral</p>	<p>Right bronchus swollen almost shut. Bifurcation 4 mm. thick. Foreign body deep in lower lobe of lung buried in granulations, nothing resembling bronchial wall around it. Pathology not due to foreign body.</p>
	<p>2 yrs.</p>	<p>Carpet tack</p>	<p>Left main bronchus 3 months 22 days</p>	<p>None</p>	<p>5 mm.</p>	<p>Buried point. Pathology 3 months' sojourn</p>	<p>Side-curved</p>	<p>Shaft, inward rotation method</p>	<p>Extraction Cure</p>	<p>2 min. 45 sec.</p>	<p>Oral</p>	<p>Point buried in median wall slightly above the lower lobe bronchus. Tack pushed downward, rotated and removed. Suppuration below tack.</p>


## HARDWARE—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 859  859	3 yrs.	Carpet tack	Left main bronchus 7 days	None	5 mm.	None	Side-curved	Shaft	Extraction Cure	4 min. 45 sec.	Oral	Head of tack in left main bronchus. Point of tack projecting above carina. Shaft of tack in wall of the right main bronchus, point of trachea in right wall of trachea at the upper edge of the orifice of the right main bronchus.
Fbdy. 974  974	6 yrs.	Carpet tack	Left lower lobe bronchus, 5 mos. in lung tissue near diaphragm, 5 days	None	5 mm.	Fbdy. in lung tissue	Side-curved	Point	Extraction Cure	11 min. 10 sec.	Oral	Large quantity of pus coming up from left lung; removed by sponge pumping. Fbdy. found at bottom of left lung almost parallel to diaphragm.
Fbdy. 907  907	2 yrs.	Bent carpet tack	Right middle lobe bronchus, 13 days	None	5 mm.	Acute stenosis	Side-curved	Bent shaft	Extraction Cure	6 min. 45 sec.	Oral	Middle lobe bronchial orifice swollen shut and containing pus and bubbles of gas or air.


<p>Fbly. 644</p> 	12 yrs.	Brass headed tack	Right lower lobe bronchus, 37 days	None	6 mm.	Shaft buried up to 3 mm. of head	Side-curved	Shaft (Inward rotation method)	Extraction 3 min. 25 sec. Cure	Oral	Localized bronchitis. Mucosal trauma not due to foreign body.
<p>Fbly. 676</p> 	3 1/2 yrs.	Brass headed tack	Right bronchus, 13 days	None	5 mm.	Disengaging point located in mucosa up to head	Side-curved	Near point (Inward rotation method)	Extraction 33 min. 20 sec. Cure	Oral	Very severe laryngotracheobronchitis. Exudate in bronchial tissue; not due to foreign body.
<p>Fbly. 677</p> 	4 yrs.	Brass headed tack	In mediastinum below bifurcation 3 mos. 2 days	None	5 mm.	Fbly. in mediastinum. Suppurative process (stenosis)	Side-curved	Shaft near point (Inward rotation method)	Extraction 9 min. 17 sec. Cure	Oral	Left cord paralyzed. Laryngeal walls thickened. Cord flattened. Subglottic swelling moderate degree. Diffuse tracheobronchitis. Right bronchus less inflamed. Left bronchus swollen almost shut below entrance of upper lobe bronchus. In ventral wall flattened. 181 cm. from upper teeth. Very little pus and secretion coming up from below the narrowed point. No. 6 body. In bronchus of a woman. Pathologically evidently not due to foreign body.

## HARDWARE—Continued


Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Fragments	Point of Suture	Result	Time	Route	Comment
Fbdy. 687 	10 yrs.	Brass headed tack	Right inferior lobe bronchus, many years	None	6 mm.	Tough granulation closing bronchus. Pus and shaft buried. Pus. Blood. Pathology from many years' sojourn	Side-curved	Point	Extraction Cure	8 min. 19 sec.	Oral	Abscess completely cicatrized and patient perfectly well at end of 6 months.
Fbdy. 694 	7 yrs.	Brass headed tack	Left inferior lobe bronchus, 3 mos. 20 days	None	5 mm.	Point buried. Pathology from 4 months' sojourn	Plain	Shaft near point (Inward rotation method)	Extraction Cure	3 min. 14 sec.	Oral	Inferior lobe bronchus swollen shut. Quantities of pus below obstructing head of tack.
Fbdy. 707 	4 yrs.	Brass headed tack	Right main bronchus, 3 days	None	5 mm.	Point and half shank buried in wall of right bronchus	Side-curved	Shaft (Inward rotation method)	Extraction Cure	3 min. 55 sec.	Oral	Posterior wall of trachea collapsed almost touching anterior wall. Right bronchus completely compressed from behind forward. (Adenopathy? Thymus?)

	9 yrs.	Brass headed tack	Slight lower lobe bronchus, 1 week	None	6 min.	Tack fixed and anchored tack	Side-curved	Shaft (Inward rotation method)	Extraction Cure	26 sec.	Oral	<p>Point of tack in orifice of upper lobe bronchus. Head in stem bronchus. Point was brought into the same axis as that of the bronchus by means of side-curved forceps. On traction being made the tack, secured quite firmly anchored by the head but was gradually disengaged and the head was drawn against the distal tube until the pus was not troublesome during bronchoscopy but after removal patient coughed up considerable quantities of pus.</p>
	5 yrs.	Brass headed tack	Right lower lobe bronchus, 5 days	None	5 min.	Buried point, transfixed shaft	Side-curved	Shaft (Inward rotation method)	Extraction Cure	3 min. 3 sec.	Oral	<p>Trachea and bronchi quite inflammatory. No sign of trauma from previous bronchoscopy. Tack found in right inferior lobe bronchus with point buried in the wall midway between the anterior and median points. None of the taper of the point visible. Point pushed downward with side-curved forceps, disengaged and brought into bronchoscope.</p>

## HARDWARE—Continued



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 917  917	9 yrs.	Brass-headed tack	Left main bronchus, 6 days	None	9 min.	Malpresentation; point upward in left main bronchus; head at center of upper lobe bronchus; tack too large to be pushed down into either upper or lower lobe bronchus rendering disengagement of point difficult	Tucker	Shaft	Extraction Cure	18 min. 19 sec.	Oral	Swelling of bronchial mucosa and diminution of lumen probably not due to foreign body. Tucker forceps invaluable in solving mechanical problem.





<p>Flody. 933</p> 	6 yrs.	Brass headed tack	Right main bronchus, about 7 mos.	None	5 min.	<p>Pathology from perforation into pleural cavity; not abscess; pyopneumothorax; esophago-pleural fistula; swallowed food coming out through intercostal wound; pleural cavity pathologic from 7 months' sojourn of tack</p>	Tucker	Shaft	Extraction 10 sec. Bronchoscopy	Onal	<p>Bronchoscopy: Subglottic and trachea, the very much swollen, the subglottic masses nearly meeting in the mid-line; just below bifurcation in the right bronchus there is a mass of granulation tissue, the size of a nut, discharging pus. Further work deemed inadvisable until after healing of esophageal fistula.</p> <p>Esophagoscopy: At about the level where the left bronchus divides, and normally, a narrow vertical slit filled with reddish tissue was visible in the esophageal wall; esophagoscope readily passed beyond it into the stomach. A nasogastric tube inserted into stomach through esophagoscope and child thus fed until perforation healed. Perforation from esophagus into pleura not due to foreign body; which was in lung.</p>
---	--------	-------------------	-----------------------------------	------	--------	--	--------	-------	---------------------------------	------	--

## HARDWARE—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
 <p>Fbly. 985</p>	8 yrs.	Brass headed tack	Right stem bronchus, 72 days	None	5 mm.	Buried point	Tucker	Stem	Extraction Cure	1 min. 47 sec.	Oral	Right stem bronchus below orifice of upper lobe bronchus appeared to be completely filled with granulations; after aspiration of pus, orifice of middle lobe bronchus could be seen on deep inspiration only; disappearing on expiration; stem of tack found buried in granulation tissue; head of tack out of sight in the granulations.
 <p>Fbly. 1003</p>	5 yrs.	Brass headed tack	Right lower lobe bronchus, 1 month	None	5 mm.	Pathology from bronchial biopsy, 1 month's duration	Side-curved and Tucker	Point and shaft	Extraction Cure	9 min. 52 sec.	Oral	Entire stem bronchus from level of middle lobe bronchus downward filled with granulation tissue and thick pus.


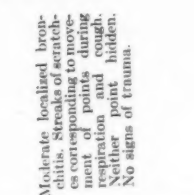
Fbdy. 648 	5 yrs.	Nail	Left main bronchus, 9 mos.	None	5 min.	Pathology from 9 months' sojourn	Side-curved	Point after disem-bedded	Extraction Cure	2 min. 27 sec.	Oral	Right bronchus normal; lower upper lobe bronchus. Left bronchus lined with granulation tissue. Point of nail buried in internal left bronchial wall below carina.
Fbdy. 654 	8 yrs.	Nail	Left bronchus, 1 year, 5 months	None	5 min.	Pathology from 1 year and 3 months' sojourn			Foreign body not found, recovered in stools	21 min. 40 sec.		Localized bronchitis in left inferior lobe bronchus but no tack. Tack evidently coughed up and swallowed after x-ray and before bronchoscopy.
Fbdy. 772 No specimen	2 1/2 yrs.	Nail	Left upper lobe bronchus, 9 mos.	None	5 min.	Upper lobe bronchus compression stenosis from mediastinal abscess		Not seized	Disl. Cardiac myopathy.	26 min. 54 sec.		Left bronchus at orifice of upper lobe bronchus filled with granulation tissue but very freely. Nail not visible anywhere but head could be felt with closed side-curved forceps around the corner in the upper lobe bronchus. Death from mediastinal abscess 24 days after bronchoscopy. (Myopathy and mediastinal abscess, 9 months' sojourn of foreign body in lung.

## HARDWARE—Continued


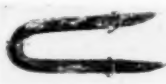
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 913 	2 yrs.	Nail	Right main bronchus, 9 days	None	3 mm.	Presentation	Tucker	Shaft (Inward rotation method)	Extraction Cure	1 min. 21 sec.	Oral	First ray showed nail in left main bronchus; second ray, 5 days later showed it to be in right bronchus. (This examination should be repeated immediately before endoscopy for verification.)
Fbdy. 921 	3 yrs.	Nail	Left main bronchus, head in left upper lobe bronchus, 10 days	None	5 mm.	Malpresentation	Tucker	Shaft	Extraction Cure	3 min. 3 sec.	Oral	




# CHARTED EXPERIENCE OF FOREIGN BODY CASES.

1023


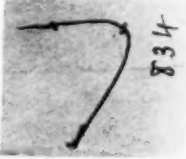
<p>Fldy. 634</p> 	<p>4 yrs.</p>	<p>Staple</p>	<p>Left main bronchus 25 days</p>	<p>None</p>	<p>5 mm.</p>	<p>Double pointed sharp staple points up in small bronchus swollen shut.</p>	<p>Rotation</p>	<p>Curved end after turning (Cephalic version, extreme-ities being turned downward, changing course and presentation for withdrawal)</p>	<p>Extraction Cure</p>	<p>18 min. 9 sec.</p>	<p>Oral</p>	<p>Bronchus swollen almost shut, proximally.</p>
<p>Fldy. 690</p> 	<p>53 yrs.</p>	<p>Staple</p>	<p>Right inferior lobe bronchus, 11 days</p>	<p>Local</p>	<p>8 mm.</p>	<p>None</p>	<p>Slightly curved</p>	<p>One point, the other point being protected by lip of tube mouth</p>	<p>Extraction Cure</p>	<p>41 sec.</p>	<p>Oral</p>	<p>Moderate localized bronchitis. Strained to move, as evidenced by coughing and points during respiration and cough. Neither point hidden. No signs of trauma.</p>

## HARDWARE—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 825 	4 yrs.	Staple	Trachea, 5 days	None	Laryngoscope	Points upward, buried in tissue below the glottis; narrow; rigid wire; very sharp points; danger of dropping deeper	Rotation	Head	Extraction Cure	1 min. 35 sec.	Oral	Points buried in swollen subglottic tissues. Posterior point readily seized and advanced up out of the larynx, the anterior point then advanced to the trachea and the curved portion was brought upward. Curved portion then forced posteriorly against membranous wall of trachea and caused to emerge by pressure exerted in a posterior direction by spatular tip of laryngoscope.
Fbdy. 975 	7 yrs.	Staple	Right stem bronchus, 26 days	None	5 mm.	Doubled-pointed tack, pointed upward; pathology and trauma	Rotation	Head; vertex of points at junction of upper lobe bronchus	Extraction Cure	39 min. 42 sec.	Oral	Found in right stem bronchus just below orifice of upper lobe bronchus. Anterior end of upper lobe bronchus and removed.

Fbdy. 661 	48 yrs.	Carpet staple	Left bronchus, 5 months	None	9 mm.	Points upward; buried in swollen mucosa	Side-curved	Head end after cephalic version (presenting extremities turned downward)	Extraction 2 min. 37 sec. Cure	Oral	Internal point buried in bronchial wall. External point hidden by granulation tissue. Localized bronchitis. Pathology slight considering length of sojourn.
Fbdy. 636 	3 yrs.	Screw	Right bronchus, 3 months	None	5 mm.	Fbdy. obscured by pathology from 3 months' sojourn	Side-curved		Extraction 25 min. 10 sec. Cure	Oral	Localized bronchitis, near foreign body; mucosa elsewhere normal.
Fbdy. 684 	3 yrs.	Screw	Right inferior lobe bronchus, 35 days	None	5 mm.	Proximal acute stenosis; granulation tissue; pus; no forceps spaces (35 days' sojourn)	Straight and side-curved	Threaded end	Extraction 2 min. 13 sec. Cure	Oral	Intense subglottic edema. Tracheitis. Mucosa inflamed or left bronchus inflamed. Intense right bronchitis. Swelling of upper lobe bronchus and middle lobe bronchus much swollen. Inferior lobe bronchus swollen almost shut. Screw found below swelling.



## HARDWARE—Continued

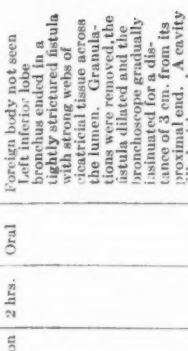

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Suture	Result	Time	Route	Comment
Case 686 	11 yrs.	Screw	Right inferior lobe bronchus, 1 yr. 6 mos.	None	6 mm.	Fibrous structure and inflammatory tissue tightly holding body; increased in size (1½ years' sojourn)	Side-curved	Point	Extraction Cure	12 min. 17 sec.	Oral	Right inferior lobe bronchus closed by pulling and granulation tissue.
Case 761 No Specimen	32 yrs.	Piece bent wire	Lingual tonsil, 2 hours	None	None	None	Alligator	One point	Extraction Cure		Oral	Bent wire embedded in lingual tonsil in front of epiglottis.
Case 834 	9 mos.	Bent wire	Hypopharynx, bent and curling through pyriform sinus, 3 days	None	Laryngoscope	None; wire soft and malleable (annealed)	Alligator	Near one end	Extraction Cure	2 min. 40 sec.	Oral	






<p>Fbdy. 678</p>  <p>678</p>	8 yrs.	Cartridge blank	Left bronchus at orifice of upper lobe; 38 days	None	6 mm.	Proximal end; stenosis; granulations; pus; no forceps spaces; 38 days' sojourn	Sile-curved	Periphery	Extraction Cure	8 min. 31 sec	Oral	Granulations bulking into left main bronchus proximally to orifice of upper lobe bronchus.
<p>Fbdy. 659</p>  <p>659</p>	8 yrs.	Umbrella tip	Bottom of right lower lobe; 5 1/2 or 6 years	None	6 mm.	Connective tissue barrier at bottom of out, containing many fistular; 6 years' sojourn	Plain	Proximal end	Extraction Cure	11 min. 45 sec.	Oral	Pus very foul after connective tissue barrier was removed. Whole lower lobe of right lung honeycombed with fistulous passages.
<p>Fbdy. 749</p>  <p>749</p>	4 yrs.	Hinge from toy	Right bronchus; 4 days	None	5 mm.	Malpresentation	Rotation	One corner, version method	Extraction Cure	6 min. 27 sec.	Oral	Subglottic tissues intensely swollen.




## HARDWARE—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes-thetic	Tube	Problem	For-ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 770 	6 yrs.	Metal pencil ferrule	Left inferior lobe bronchus, upper edge occluding entirely orifice of upper lobe bronchus. 5 days	None	5 mm.	Tight impaction. Hollow body. No external creases or spaces.	Side-curved	By edge, one blade of forceps inside, opposite edge covered by lip of tube—mouth drawing with—drawal to protect tissues	Extraction Cure	5 min. 22 sec.	Oral	Tight impaction, double less due to atelectatic condition of that lung consequent upon complete occlusion of the bronchus by the body. Absorption of air led to negative pressure drawing foreign body downward.
Fbdy. 785 	3 yrs.	Hook-shaped object	Esophagus partly above partly below crico-pharyngeus. 10 days	None	45 cm. esoph. ago-	Sharp double hook. One hook above crico-pharyngeus, preventing movement downward, with hook below crico-pharyngeus preventing downward movement. Very large object for size of esophagus. Dyspnea.	Side-curved	Lower hook after embedding	Extraction Cure	9 min. 37 sec.	Oral	Fbdy. covered with food, blood-stained secretions and granulations. Intense dyspnea super-vened. Hook was approached with the esophageal speculum. Localized esophagitis.





<p>Fbdy. 783</p> 	<p>28 yrs.</p>	<p>Metal pencil top with eraser attached</p>	<p>Left lower lobe bronchus 1 1/2 yrs.</p>	<p>Local</p>	<p>Fibrous tissue hard.</p>	<p>Side-curved</p>	<p>Extraction</p>	<p>2 hrs.</p>	<p>Oral</p>	<p>Foreign body not seen. Left inferior lobe bronchus ended in a tightly strictured isthula with strong webs of granulation tissue across the lumen. Granulations were removed, the isthula dilated and the bronchoscope gradually insinuated for a distance of 3 cm. from its extremity. The cavity was filled with mushy granulations was found, and in a small portion of the foreign body was discovered. The foreign body was manipulated and its proximal end caught in the stricture and was removed the third time it was grasped.</p>
<p>Fbdy. 837</p> 	<p>2 mos.</p>	<p>Hair pin</p>	<p>In hypopharynx and orifice of esophagus about 1 month</p>	<p>None</p>	<p>Both points upward and buried in mucosa</p>	<p>Side-curved. (Tucker forceps better, but not then devised)</p>	<p>One point, after other point bent inward</p>	<p>Extraction</p>	<p>1 min. 50 sec.</p>	<p>Oral</p> <p>Child was rayed on suspicion of enlarged thymus because of stridorous respiration of one month's duration.</p>




## HARDWARE—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 843 	6 yrs.	Piece lead pipe	Left upper lobe bronchus, protruding into main bronchus, 4 days	None	6 mm.	Very small forceps spaces	Sider-curved	Over outside of presenting end	Extraction Cure	2 min. 28 sec.	Oral	
Fbdy. 930 	37 yrs.	Metallic object (nature unknown)	Right lower lobe bronchus, 35 years (about)	Local		Patient moribund from cachexia			Foreign body removed post-mortem	47 min. 59 sec.	Oral	On bronchoscopy fistula found communicating with right interior lobe bronchus. Bronchoscopy postponed to next day. Feeble patient rest; death 4 days after bronchoscopy from pulmonary hemorrhage. Autopsy revealed epithelioma (Metaplasia). See report of W. J. Jackson, Trans. A.M.A., Feb. 18, 1922.
Fbdy. 968 	6 yrs.	Paper fastener	Right stem bronchus below orifice of upper lobe bronchus, 8 days, points up	None	5 mm.	Diverging points	Plain	Both points compressed together	Extraction Cure	1 min. 9 sec.	Oral	



Fbdy. 1009  1009	3 yrs.	Umbrella-headed tack	Left main bronchus 3 days	None	5 mm.	Buried point; Mushroom anchor; swollen mucosa	Tucker	By point	Extraction Cure	4 min. 17 sec.	Oral	Bronchus filled above tack with thick pus, looking like exudate. All bronchial segments were very much inflamed.
Fbdy. 1011 No Specimen	2 yrs. 5 mos.	Screw	Left bronchus 1 cm. below bifurcation 10 days	None	5 mm.	Presentation and proper grasping	Tucker	By point	Extraction Cure	2 min. 43 sec.	Oral	
Fbdy. 1041  1041	8 yrs.	Ball bearing ball	Right inf. lobe bronchus below orifice middle lobe bronchus. 16 days	None	7 mm.	Ball tightly impacted	Plain with cupped faces	Poles	Extraction Cure	23 min. 10 sec.	Oral	Mucosa swollen, proximally almost closing lumen. Swollen mucosa pressed backward with tube mouth and foreign body seized.
Fbdy. 1054  1054	27 yrs.	Pencil cap	Left lower lobe bronchus. 21 yrs.	Local	7 mm.	Frangible foreign body in abscess cavity filled with fibrous granulations and pus	Plain	Edge	Extraction Cure	33 min. 20 sec. 28 min. 35 sec.	Oral	Right bronchial orifice inflammatory; left bronchus chronically inflamed; mucosa thickened. Just below left upper lobe bronchus stem per lobe bronchus stem completely obliterated. Forceps gradually insinuated through organized tissue. Below narrowing segment cavity filled with granulation tissue in which foreign body was located encased in capsule of fibrous tissue. Foreign body crumbled at touch of forceps. Mass removed. One fragment embedded in tissue; not torn loose. Removed at second bronchoscopy.

## HARDWARE—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1050  1056	3 yrs. 8 mos.	Tack	Right stem bronchus. 5 months	None	5 mm.	Buried point; anterior anterior; pus; bleeding granulations	Tucker	By point	Extraction Cure	4 min. 40 sec.	Oral	Right main bronchus filled with granulations; quantity of pus came up from below as soon as tack was touched.
Fbdy. 1061  1061	4 yrs.	Screw	Right lower lobe bronchus. 5 days	None	5 mm. aspirating	Presentation and proper grasping	Tucker	By point	Extraction Cure	7 min. 22 sec.	Oral	
Fbdy. 1093  1093	3½ yrs	Carpet tack	Right main bronchus. 4 days	None	5 mm.	Buried tack; transfixed	Tucker	Near head, point trailing	Extraction Cure	2 min. 37 sec.	Oral	Version done at orifice of upper lobe bronchus.
Fbdy. 1095  1095	3 yrs.	Screw	Left bronchus above orifice of left upper lobe. 1 yr. 9 mos.	None	5 mm. aspirating	Proper presentation and grasping; bleeding granulations; profuse flow of pus	Side- curved	Pointed end	Extraction Cure	3 min. 16 sec.	Oral	Left bronchial orifice filled with exuberant bleeding granulations, buried in which the foreign body was found.

 Fbdy. 1104 1104	12 yrs.	Nail	Lower portion left upper lobe. 10 years	None	6 mm. aspirating	Location of foreign body in left upper lobe	Plain	Pointed end	Extraction Cure	22 min. 22 sec.	Oral	Nail embedded in pathologic tissue consisting of fistulae, fibrous bands, exuberant granulations in the lower portion of left upper lobe.
 Fbdy. 1117 1117	14 yrs.	Umbrella-headed tack	Right middle lobe bronchus. 2 yrs. 2 mos.	None	6 mm. aspirating	Foreign body bedded in granulations below bronchial stricture	Tucker	Near point, after dilatation of stricture	Extraction Cure	13 min. 20 sec.	Oral	Stem bronchus narrowed below the middle lobe bronchus; below the narrowing the bronchus widened into a bronchiectatic cavity deep down in which was a mass of granulations and a tack which the tack was buried.
 Fbdy. 1148 1148	18 yrs.	Umbrella-headed tack	Right main bronchus. 9 yrs.	None	7 mm.	Corroded flexible tacks bedded in granulations below tightly strictured bronchus	Tucker	Point after dilatation of stricture; head remained and was seized flatwise	Extraction Cure	7 min. 50 sec.	Oral	Right bronchial orifice very much narrowed, lumen of orifice being about 3 mm. in diameter with a mass of granulations filling the lower part of the lumen from view. After dilatation point of tack found in contact with anterior wall just below narrowest part of stricture. Stem of tack embedded under covers and was removed in fragments; head then removed.



## JEWELRY

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 718 	6 yrs.	Portion of collar button	Left main bronchus. 1 yr.	None	5 mm.	None	Side-curved	Presenting end	Extraction Cure	3 min. 13 sec.	Oral	Nodule apparently granulomatous hanging on prominent side of foreign body.
Fbdy. 748 	21 yrs.	Open bar pin	Left lower lobe bronchus. 18 yrs.	Local	7 mm.	Fibrous stenosis	Side-curved	Point	Extraction Cure	9 min. 14 sec.	Oral	Opening of abscess cavity leading off posteriorly from inferior lobe bronchus was discovered with the aid of Dr. Manges' anoscopic guidance. After passing the lip of the bronchus, the hook could be inserted in this orifice. The point was rotated with forceps until the point was protected by the lip of the bronchoscope. The point then slid along the inner canal permitting withdrawal of the pin through bronchoscope. Latter withdrawn afterward. Pin very much corroded and broke in the hand after removal.



<p>Fbdy. 954</p>  <p>954</p>	<p>9 mos.</p>	<p>Collar button</p>	<p>Esophagus below crico-pharyngeus, 9 days</p>	<p>None</p>	<p>7 mm.</p>	<p>Malpresen- tation</p>	<p>Alliga- tor cupped</p>	<p>Post. after version</p>	<p>Extraction Cure</p>	<p>4 min. 10 sec.</p>	<p>Oral</p>	<p>Collar button jammed be- low cricopharyngeus and wedged into the posterior wall of the trachea below the cricoid; tracheal lu- men noted beneath the mass appeared to be bulged for- ward, especially on its right posterior quadrant. Mass of cotton about 3 cm. long and 5 mm. in diameter removed from above the collar button. No bleeding or ulceration in both anterior and poster- ior walls of esophagus. Post. presented by version and grasped with alliga- tor forceps and removed.</p>
<p>Fbdy. 979</p>  <p>979</p>	<p>6 yrs.</p>	<p>Collar button</p>	<p>Esophagus just below crico- pharyngeus, 5 days</p>	<p>None</p>	<p>7 mm.</p>	<p>Malpresen- tation</p>	<p>Sile- curve.</p>	<p>Post. after version</p>	<p>Extraction Cure</p>	<p>5 min. 20 sec.</p>	<p>Oral</p>	<p>Collar button lying in slit in posterior esophageal wall.</p>
<p>Fbdy. 1051</p>  <p>1051</p>	<p>13 mos.</p>	<p>Ring</p>	<p>Esophagus 11 mos. 3 weeks</p>	<p>None</p>	<p>5 mm.</p>	<p>Presen- tation; thymic compres- sion; in- flamma- tion; ulceration</p>	<p>Mos- quito</p>	<p>Presenting part</p>	<p>Extraction Cure</p>	<p>20 min. 56 sec.</p>	<p>Oral</p>	<p>Both trachea and eso- phagus seem to be com- pressed by a ring of mus- cle, the trachea which threatens asphyxia every time the esophagoscope is introduced to the vicini- ty of the foreign body. Foreign body not visible. Esophagus not seen. Esophagus dilated by masses coming in from all sides and covered with granu- lulation tissue. On dis- placing these folds the small portion of the upper edge of ring was visible.</p>


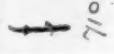
## JEWELRY—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Exposure	Point of Seizure	Result	Time	Route	Comment
1086  1086	7 yrs.	Collar button	Right lower lobe bronchus. 8 mos.	None	6 mm.	Moribund patient with one lung completely collapsed and $\frac{1}{4}$ of other lung out of function from suppuration	Plain	Post	Extraction Death	14 min. 17 sec.	Oral	Patient admitted with left pyopneumothorax. At bronchoscopy collar button found in an area of right lower lobe bronchus down in right lower lobe. Patient had one lung collapsed and at least $\frac{1}{4}$ of the other lung out of service, leaving only $\frac{3}{4}$ of one lung with which to breathe. Patient kept alive on table by oxygen through bronchoscope. Intercostal drainage, left side 5 days after bronchoscopy. Death one week later.
1105  1105	4 yrs.	Beauty pin	Left bronchus 1 cm. below bifurcation. 23 days.	None	5 mm.	Open bar pin	Sideways	By pointed branch	Extraction Cure	12 min. 37 sec.	Oral	Point-protected method of removal.






# PINS AND NEEDLES

## CHARTED EXPERIENCE OF FOREIGN BODY CASES.

1037

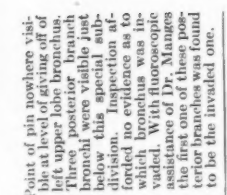
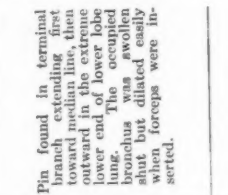
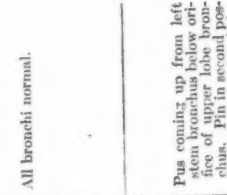
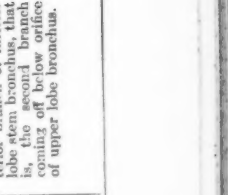
Fbody. 635	19 yrs.	Shawl pin	Right inferior lobe bronchus. 7 days	Local	7 mm.	Point buried in bronchial wall	Side- curve.	Near point Inward rotation	Extraction Cure	14 min. 31 sec.	Oral	No sign of trauma or slightest injury from pre- vious bronchoscopy.
												
Fbody. 630	3 yrs.	Shawl pin	Left inferior lobe bronchus. 21 hours	None	5 mm.	Disenag- ing point		Near point Inward rotation	Extraction Cure	4 min. 17 sec.	Oral	
												
Fbody. 662	20 yrs.	Pin	Pharynx. 1 day	None	Laryn- goscopa	None		Near point	Extraction Cure	48 sec.	Oral	
												
Fbody. 710	40 yrs.	Pin	Middle lobe bronchus. 5 days	Local	7 mm.	Transfixed crosswise						Died of hemorrhage.
												

## PINS AND NEEDLES—Continued






Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 712  Fbdy. 715  712	50 yrs.	Pin	Esophagus level 7th cervical vertebra. 4 days	None	9 mm.	Pin buried up to head in anterior esophageal wall		Point	Extraction Cure	3 min. 17 sec.	Oral	
Fbdy. 726  715	31 yrs.	Shawl pin	Left upper lobe bronchus. 16 days	Local	7 mm.	Pin around corner in anterior branch of left upper bronchus. Point buried		Point inward rotation	Extraction Cure	12 min. 4 sec.	Oral	
Fbdy. 733  726	41 yrs.	Glass headed shawl pin	Inferior lobe of right lung	Local	7 mm.	Disengaging point from electrical tissue behind the overhanging of stricture	Slide-curved and plain	Near point inward rotation	Extraction Cure	3 min. 25 sec.	Oral	Right bronchus ulcerated, narrowing down into a funnel-shaped stricture. Pinsurrounded by brownish material which Stensen's duct was in contact with. Stricture was so narrow that the head engaged, but it was pulled through without great difficulty.
Fbdy. 733  733	20 yrs.	Pin (Common)	Right lower lobe bronchus. 3 days	Local	5 mm.	Finding pin in a minute branch bronchus too small for Stensen's chroscope to enter	Plain	Point	Extraction Cure	35 min.	Oral	No abnormality visible anywhere. Fluorescopic assistance of Dr. Manges required for identification of invaded bronchus.





# CHARTED EXPERIENCE OF FOREIGN BODY CASES.

1039

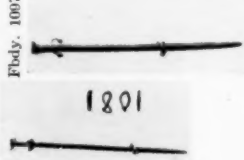
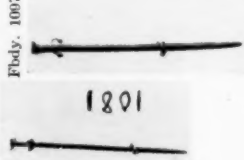

	2 yrs.	Pin (common)	Left lower lobe bronchus. 9 days	None	5 mm.	Small pin in tiny branch bronchus	Plain	Point	Extraction Cure	30 min. 21 sec.	Oral	Point of pin nowhere visible at level of giving off of left upper lobe bronchus. Three posterior branch bronchi were visible just below this apical subdivision and appeared to form a loop of evidence as to which bronchus was invaded. With fluoroscopic assistance of Dr. Manges the first one of these posterior branches was found to be the invaded one.
	15 yrs.	Pin (common)	Extreme lower end of right lower lobe bronchus. 1 month	Local	5 mm.	Extreme depth of pin in a minute bronchus, leading from a small branch "around the corner"	Side-curved	Point	Extraction Cure	17 min. 34 sec.	Oral	Pin found in terminal branch extending first toward middle of lower end of lower lobe lung. The occupied bronchus was swollen shut but dilated easily when forceps were inserted.
	2½ yrs.	Sewing machine needle	Right lower lobe bronchus. 1 month	None	5 mm.	None	Side-curved	Point	Extraction Cure	5 min.	Oral	All bronchi normal.
	2 yrs. 9 mos.	Pin (common)	Left lower lobe bronchus. 18 days	None	5 mm.	Finding the invaded bronchus	Side-curved	Point	Extraction Cure	8 min. 35 sec.	Oral	Pin coming up from left stem bronchus below orifice of upper lobe bronchus. Pin entered lower branch of inferior lobe stem bronchus, that is, the second branch coming off below orifice of upper lobe bronchus.

## PINS AND NEEDLES—Continued



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 847  Fbdy. 851 No Specimen 	33 yrs.	Shawl pin	Right main bronchus. One week	Local	7 mm.	Point of pin at orifice of middle lobe bronchus with point in point in radial wall of stem bronchus buried about 1 cm.	Side-curved	Point	Extraction Cure	33 min. 27 sec.  2 min. 5 sec.	Oral	
Fbdy. 860  Fbdy. 869  	14 mos.	Pin (common)	Left lower lobe, in lung tissue, about 5 months	None		Extreme depth of pin in lung of so small a child, pin removed in lung tissue, not in a bronchus			Pin coughed up following two attempts at removal during which pin was moved to a higher level	21 min. 5 sec.		Subglottic tissue found swollen. Pin at level of diaphragm. Tissue barrier quite firm. At second bronchoscopy point-end of pin found buried in tissue to a distance of about 6 mm.
	23 yrs.	T-shaped pin (paper fastener)	Head in right bronchus, point sticking in left tracheal wall just above carina, 5 days	Local		Point buried. Transfixed	Side-curved	Near point. Inward rotation	Extraction Cure	45 sec.	Oral	Mass of thick, tough, grey secretion covering the portion of pin that had buried itself in tracheal mucosa.
	14 yrs.	Pin (common)	Right lower lobe bronchus, 33 days	None	5 mm.		Side-curved	Near point. Inward rotation	Extraction Cure	23 min. 17 sec.	Oral	Pin found in a tiny branch bronchus, orifice of which was swollen shut.

Fbdy. 912		12 yrs.	Pin (common)	Point in left pyriform sinus; head in right pyriform sinus, 1 day	None	Esoph-ageal spec-ulum	Transfixed	Mathieu	Near point. Inward rotation	Extraction Cure	2 min. 52 sec.	Oral
Fbdy. 986		10 yrs.	Shawl pin	Left bronchus, 48 days	None	5 mm.	Pin in small peribronchus of a child	Tucker	Point	Extraction Cure	11 min. 28 sec.	Oral
Fbdy. 1055		45 yrs.	Needle	Pharynx, point upward, 6 days	Local	Laryngo-cope	Presenta-tion and preven-tion of esophagus downward	Papil-loma	Uppermost end	Extraction Cure	1 min. 30 sec.	Oral
Fbdy. 1002		41 yrs.	Pin (common)	Esophagus below crico-pharyngeal folds, point downward, 3 hours	None	Forbes spec-ulum	Presenta-tion and preven-tion of puncture by over-riding	Tucker	Shaft be-low head	Extraction Cure	36 sec.	Oral


## PINS AND NEEDLES—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1081 	25 yrs.	Pin (common)	Pharynx, about 2 hours	None			Angular nasal		Extraction Cure		Oral	
Fbdy. 1097 	12 yrs.	Pin (common)	Hypopharynx, point downward, 4 days	None	Hypopharyngoscope	Presentation, avoidance of over-riding and loss downward	Alligator (Moshier's)	Shaft below head	Extraction Cure		Oral	Pin lying across larynx, point embedded in right pharyngeal wall, posteriorly, the head end directed toward base of epiglottis, left side.
Fbdy. 1103 	17 yrs.	Pin (common)	Right lower lobe bronchus, point upward, 28 days	Local	6 mm.	Finding pin located, presenting enlwise in tiny anterior branch of inf. lobe bronchus in periphery of lung	Plain	Pointed end	Extraction Cure	13 min. 35 sec.	Oral	


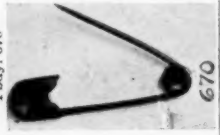






 <p>Fbdy. 1151</p>	18 yrs.	Pin (common)	Larynx. 13 hours	None	Laryngoscope	Prevention of lodgment downward	Tucker	Shaft near pointed end	Extraction 35 sec. Cure	Oral	Pin found with head in right ventricle near anterior commissure, point embedded in posterior surface of epiglottis. Patient in Jackson position for removal.
 <p>Fbdy. 1075</p> <p>1151</p> <p>1075</p>	19 yrs.	Shawl pin	Left main bronchus	Local	7 mm.	Long, pointed body.	Tucker	Near point	Extraction 1 min. 19 sec. Cure	Oral	

SAFETY PINS



 <p>Fbdy. 651</p> <p>651</p>	11 wks.	Safety-pin open	Upper esophagus level of larynx, 1 day	None	None	Straight	Spring end	Extraction Cure	Oral	
---	---------	-----------------	--	------	------	----------	------------	-----------------	------	--

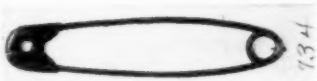


## SAFETY PINS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- eign body	Point of Seizure	Result	Time	Route	Comment
Fbdy. 660 	15 mos.	Safety-pin open	Right inferior lobe bronchus, 10 days	None	4 mm.	Open safe- ty-pin in a small bronchus point up	Side- curved	Pointed branch	Extraction Cure	7 min. 51 sec.	Oral	Considerable degree of infectious perichondritis and broncho-pneumonia, localized.
Fbdy. 670 	6 yrs.	Safety-pin open	Trachea, 4 days	None	5 mm.	Point up	Side- curved	Pointed branch	Extraction Cure	7 min.	Oral	
Fbdy. 695 	12 mos.	Safety-pin open; point down	In stomach two months. Regurgi- tated into esophagus	None	7 mm.	Prevention of over- riding, point be- ing down- ward and having already perforated	Side- curved	Spring end				



 <p>Fbdy. 697</p>	32 yrs.	Safety-pin open	Left bronchus, 15 days	Local	7 mm.	Point upward	Side-curved	Pointed branch	Extraction Cure	4 min. 49 sec.	Oral	Localized bronchitis, left; left bronchial orifice not stenosed.
 <p>Fbdy. 701</p>	9 mos.	Safety-pin open	Trachea 1 day	None	5 mm.	None	Side-curved	Pointed branch	Extraction Cure	7 sec.	Oral	Acute laryngotracheitis, safety-pin in trachea immediately subglottic.
 <p>Fbdy. 713</p>	8 mos.	Safety-pin open	Esophagus at hiatus esophageus 1 day	None	7 mm.	Open safety-pin jammed in cardia			Pin pushed into stomach. Recovered in stools	12 min. 15 sec.		



## SAFETY PINS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 717 	7 mos.	Safety-pin open	Overhanging larynx. Point up, 1 day	None	Direct laryngoscope	Open, point up. Embolized up to spring		Pointed branch	Extraction Cure	1 min. 25 sec.	Oral	
Fbdy. 731 	10 mos.	Safety-pin open	Esophagus, 3 days	None	Esophagoscaphum	Point up	Alligator	By keeper. Pointed in tube mouth	Extraction Cure	16 sec.	Oral	Safety-pin below cricopharynx.


Fbdy. 731		18 mos.	Safety-pin closed	Trachea 8 days	None	5 mm.	None	Spring end	Extraction Cure	1 min. 10 sec.	Oral	Marked glottic and sub-glottic swelling, intense laryngotracheitis.
Fbdy. 742		7 mos.	Two safety-pins (closed and linked together)	Trachea, 8 days	None	5 mm.	Extremely large size of foreign body, extremely small size of air passages which seemed almost closed even for a 7 mos. baby.	Keeper end	Extraction Cure	2 min. 17 sec.	Oral	The upper pin seized with forceps and the pins withdrawn linked together "in a chain."
Fbdy. 747		3 mos.	Safety-pin open	Esophagus, 2 days	None	Laryngoscope	Pointed end buried in left esophageal wall up to spring.	Keeper end	Extraction Cure	45 sec.	Oral	Keeper end of pin about 4 cm. below glottis. Pin disimpacted by pushing down and pin then rotated to lip of laryngoscope.

## SAFETY PINS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 758 	20 yrs.	Safety-pin open	Left upper lobe bronchus, 6 days	Local	7 mm.	Point upward	Slider-curved	Point	Extraction Cure	2 min. 4 sec.	Oral	Pin found in left lung, spring end in upper lobe bronchus, the pin cross-wise. Point of pin drawn into bronchoscope and withdrawn through bronchoscope. Pin was bent by distal point and side outlet at proximal end of bronchoscope.
Fbdy. 766 	9 mos.	Safety-pin open	Esophagus lower end just above hiatus, 8 days	None		Pointed branch visible only for a distance of about 5 inches from spring, balance of pointed branch being buried in this tissue. Compression atension from mediastinal emphysema. Hemorrhage and gastric straightening against tube-mouth.		Keeper end	Extraction	8 min. 16 sec.	Oral	Death 7 days following removal, from broncho-pneumonia and mediastinitis.

	15 yrs.	Safety-pin open	Right main bronchus, 3 weeks	None	Point up	Side-curved	Spring end	Extraction Cure	5 min. 3 sec.	Oral	Intense tracheobronchitis. Mound of granulation tissue springing from internal wall of right bronchus. Head of pin found just below mound. Spring end not anchored. Spring too large to draw into any bronchoscope that could be safely introduced. Brought up very carefully. Did not meet with any resistance.
	4 yrs.	Safety-pin open	Larynx, point up, 3 days	None	Point up	Alligator	By keeper	Extraction Cure	25 sec.	Oral	Keeper of safety-pin hanging on the vocal cords, obscured by the overhanging of the ventricular band of the larynx. Pin seized during inspiration and withdrawn, point sheathed with the laryngoscope.
	18 yrs.	Safety-pin open	Right lower lobe bronchus. Point up. 1 yr. 10 mos.	Local	Corroded pin, open	Side-curved	Pointed branch	Extraction Cure	1 min. 35 sec.	Oral	Keeper end of pin found at orifice of abscess cavity. Foreign body rotated and removed with side-curved forceps.


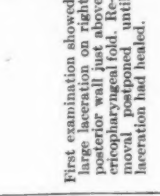

## SAFETY PINS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fldy. 806 	6 mos.	Two safety-pins (closed and linked together)	Stomach, 4 weeks	None	7 mm.	Presenting of proper part for safe withdrawal.		One of the keepers	Extraction Cure	26 min.	Oral	Pins were wrapped in folds of the stomach wall and both walls and pins were in constant motion. There were many opportunities to seize the pins during the course of the gastroscope, but each time the pins were in an inaccessible portion. I had decided upon one of the keepers as the most favorable part of the double foreign body to seize and it required the full time to get this done, so that one of them would present the keeper end for traction. Drawing gently on this one the second pin formed a second link to the chain during withdrawal. Great care was exercised in extracting the keeper end of the second pin through the hiatus and later past the cricoid.






CHARTED EXPERIENCE OF FOREIGN BODY CASES.

1051




 <p>Fbdy. 813</p>	<p>2 yrs.</p>	<p>Safety-pin open</p>	<p>Esophagus below cricopharyngeus point up, about 8 hours</p>	<p>None</p>	<p>7 mm.</p>	<p>Pointed branch of pin curved up to the spring</p>	<p>Side-curved</p>	<p>Pointed branch</p>	<p>Extraction Cure</p>	<p>1 min.</p>	<p>Oral</p>	
 <p>Fbdy. 836</p>	<p>7 mos.</p>	<p>Safety-pin open</p>	<p>Esophagus just below cricopharyngeus, 11 days</p>	<p>None</p>	<p>5 mm.</p>	<p>Open, point up. Covered with wool</p>	<p>Side-curved</p>	<p>Pointed branch</p>	<p>Extraction Cure</p>	<p>50 sec.</p>	<p>Oral</p>	<p>First examination showed large laceration on right posterior wall just above cricopharyngeal sphincter. Laceration closed until laceration had healed.</p>
 <p>Fbdy. 850</p>	<p>24 yrs.</p>	<p>Safety-pin open, point downward</p>	<p>Esophagus, 12 days</p>	<p>None</p>	<p>9 mm.</p>	<p>Entire pin except keeper buried in mucosa</p>	<p>Side-curved</p>	<p>Pointed branch</p>	<p>Extraction Cure</p>	<p>3 min. 2 sec.</p>	<p>Oral</p>	





## SAFETY PINS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 882 	1 yr.	Safety-pin caught point downward	Esophagus just above diaphragm, 4 days	None	7 mm.	Open point up.	Sides-curve.	Spring end	Extraction Cure	2 min. 33 sec.	Oral	Spring end of pin encountered just above the diaphragm and the pointed branch outward to the left buried out of sight from a point about 1 cm. distant from the spring keeper out of sight behind the esophagus. Pin closed by drawing into tube; tube-mouth protecting the esophagus from trauma by the sharp, hook-shaped keeper end of the pin.
Fbdy. 881 	2 yrs.	Open safety-pin	Larynx, point downward, 8 mos.	None	Laryngoscope	None	Alligator	Spring	Extraction Cure	3 min. 30 sec.	Oral	Child referred for treatment of laryngeal stenosis for which he was warbled with a tracheotomy tube. Routine ray examination showed foreign body in larynx. On direct laryngoscopy, pin found deeply impacted in granulation tissue which completely filled larynx. Tracheotomy had been done 8 months previously for supposed diphtheria.
Fbdy. 883 	9 mos.	Open safety-pin	Nasopharynx, 2 days	None	Laryngoscope	None.	Alligator	Spring end	Extraction Cure	40 sec.	Oral	Palate lifted with pillar retractor.

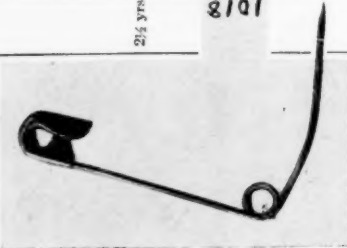

 <p>Fbdy. 889</p>	17 mos.	Open safety-pin	Esophagus 2 cm. below cricoid cartilage, 2 days point downward	None		Point up	Alligator	Pointed branch. Pointed end sheathed method.	Extraction Cure	1 min. 15 sec.	Oral	
 <p>Fbdy. 899</p>	18 mos.	Open safety-pin	Trachea 7 months, point upward	None		Point up	Alligator	Near point	Extraction Cure	1 min.	Oral	<p>Pin not visible except during inspiration; subglottic tissues swollen until they almost met in the median line below the cords. Croupiness evidently due to edema of the tissues between the cords, but to the swelling of the cords and subcordal tissues.</p>
 <p>Fbdy. 929</p>	15 mos.	Open safety-pin	Esophagus just above crossing of left bronchus, point up, 6 days	None	Esophageal specimen	Pointed branch end to end and buried for half its length in the right esophageal wall at an angle which indicated perforation	Alligator	Keeper end pointed branch pulled down and rotated into spatular end of esophageal specimen	Extraction Cure	1 min. 40 sec.	Oral	




## SAFETY PINS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 942 	9 mos.	Open safety-pin	Esophagus just above hiatus, 13 days	None	7 mm.	Point up. Pin pushed in to stomach, straightened and brought out through tube.		Endo-gastric straightening	Extraction Death	6 min. 31 sec.	Oral	Death 10 days following removal from septic pneumonia.
Fbdy. 951 	6 mos.	Open safety-pin	Esophagus 4 cm. below esophagus, 5 days. Point up	None		Point up. Small baby.	Side-curved	Pointed branch	Extraction Cure	2 min. 46 sec.	Oral	
Fbdy. 981 	2 mos.	Safety-pin open	Esophagus below crossing, left bronchus, 2 days	None	5 mm.	Point up. Removed by endo-gastric version.	Pushed into stomach with side-curved retractor, removed with rotation forceps		Extraction Cure	6 min. 35 sec.	Oral	Removed by endogastric version.

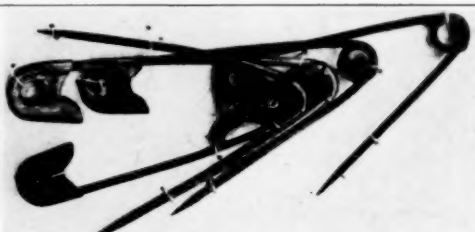

Fbdy. 934 	10 mos.	Safety-pin open	Hypopharynx, 2 days. Above cricopharyngeus	None	7 mm.	Pointed branch buried. Point up	Alligator	Point after dismembering	Extraction Cure	2 min. 53 sec.	Oral	It is very rarely that a safety pin lodges as this one did, above the cricopharyngeal fold.
Fbdy. 990 	8 1/2 mos.	Safety-pin open	Hypopharynx, above cricopharyngeus, 2 days	None	7 mm.	Point up		Spring end	Extraction Cure	15 sec.	Oral	Lodgment above cricopharyngeus is very rare.
Fbdy. 992 	10 yrs.	Safety-pin open	Larynx, 22 days	None	Laryngoscope	Pointed branch buried in cricoid cartilage posteriorly. Point up		Spring end	Extraction Cure	3 min. 52 sec.	Oral	
Fbdy. 1003 	5 yrs.	Open safety-pin	Larynx, point upward, 5 months	None	Laryngoscope	Prevention of loss downward; avoidance of trauma	Tucker	Pointed branch	Extraction Cure	39 sec.	Oral	Larynx swollen. Color putrid. Pin found in sagittal plane. Point situated method of removal.

## SAFETY PINS—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Foci	Point of Suture	Result	Time	Route	Comment
 Fbdy. 1018	2½ yrs.	Open safety-pin	Keoper in right pyriform sinus, spring cut in wall of esophagus below cricopharyngeus, point up, 3 days	None	Laryngoscope	Point embedded in wall of left pyriform sinus. Wide spread of pin	Rotation	Keoper	Extraction Cure	2 min. 45 sec.	Oral	Pin located in pharynx. Dropped to lower level during direct examination. Pin was removed 2 days later.
 Fbdy. 1039	17 mo.	Open safety-pin	Esophagus above aorta, 1 day	None	Esophagoscope and laryngoscope	Open safety pin, point upward	Side-curved and laryngeal grasping	Pointed branch	Extraction Cure	5 min. 45 sec.	Oral	Point-sheathed method of removal.

Fbdy. 1040		3 yrs.	Open safety-pin	Esophagus, buried in cricopharyngeal fold, 2 days	None	7 mm.	Open safety pin, point upward	Tucker	Pointed branch	Extraction Cure	3 min. 40 sec.	Oral	Point-sheathed method of removal.
Fbdy. 1058		6 yrs.	Open safety-pin	Right bronchus, 10 days	None	6 mm.	Open safety pin	Rotation	Pointed branch near ring	Extraction Cure	1 min. 38 sec.	Oral	Removal after version.
Fbdy. 1066		8 mos.	Open safety-pin	Esophagus below crossing of left bronchus, 1 day	None	6 mm.	Point embedded up to spring end in left esophageal wall	Rotation and Tucker	Pointed branch	Extraction Cure	9 min. 33 sec.	Oral	Withdrawn with point in tube and kept outside. Point-sheathed method.




## SAFETY PINS—Continued





Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Case 1071 	9 mos.	Four open safety pins	Esophagus, points up, over 5 weeks	None	6 mm.	Four open, upward pointed, interlocked safety-pins	Tucker; Arrow-smith pin closer	One by point; one by spring end after sneezing point.	Two pins removed esophagoscopically; 19 min. two recovered in stomach after disentanglement and gastric emplacement	36 min. 38 sec. 24 min. 19 min. 32 sec.	Two pins orally	Pins disentangled by detaching two lowermost and placing them in the stomach; two uppermost (larger and stiffer) removed personally by pin snare, and one by Tucker forceps. Arrow-smith closer used to approximate keeper and pointed branches.
Case 1073 	10 mos.	Open safety-pin	Right inferior lobe bronchus; point up, 28 days	None	5 mm.	Open safety-pin point up in infant	Side-curved	By point	Extraction Cure	12 min. 49 sec.	Oral	Pin removed by straightening against tube mouth







 <p>Fbdy. 1076</p>	7 mos.	Open safety-pin	Pharynx, 3 days	None	Laryngoscope	Open safety-pin, point up; infant	Alligator Mosher	Keeper	Extraction Cure	2 min. 10 sec.	Oral	Point of pin embedded in right wall of hypopharynx; spring extended down behind cricoid. Point snapped into laryngoscope.
 <p>Fbdy. 1084</p>	10 mos.	Open safety-pin	Esophagus, lower end, 20 days	None	3 mm.	Open safety-pin, point up, in infant	Rotation	Coil spring, endo-gastric version	Extraction Cure	4 min. 4 sec.	Oral	Endogastric version and closure of pin.
 <p>Fbdy. 1085</p>	46 yrs.	Open safety-pin	Right main bronchus, 36 years	Local	9 mm. aspirating	Open safety-pin, point up, in bronchus; pin much corroded and friable; granulations, pus	Side-curved	Pointed branch	Extraction Cure	19 min. 17 sec.	Oral	Pin found buried in granulation tissue which, with the aid of a small endotracheal catheter, excluded the orifice on expiration; on inspiration two small air spaces opened up by waiting of granulations. Large amount of pus aspirated. Pin moved in place; very much corroded. Very protuberant granulations removed with forceps. After removal of pin right bronchus found to be lined with granulation tissue down as far as middle lobes; trachea also lined with granulation tissue above bifurcation. Bronchial walls rigid.

## SAFETY PINS—Continued

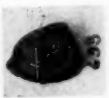
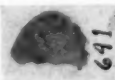


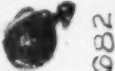
Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Suture	Result	Time	Route	Comment
 Fbdy. 1092	7 yrs.	Open safety-pin	Larynx, 2 days	None	Anterior commissure laryngoscope	Avoidance of trauma and prevention of loss downward	Alligator	Keeper	Extraction Cure	32 sec.	Oral	Pin embedded in larynx with point buried in colloid membrane. Rotated to disengage point and removed with point on spatular tip of laryngoscope.
 Fbdy. 1114	18 mos.	Closed safety-pin	Larynx and trachea, 2 days	None	Laryngoscope	None	Alligator	Spring end	Extraction Cure	40 sec.	Oral	
 Fbdy. 1111	3 yrs.	Open safety-pin	Esophagus behind heart, 2 days	None	Gastroscopy	Large opening in point; avoidance of trauma. Child	Rotation	Coll-spring end	Extraction Cure	20 min. 22 sec.	Oral	Removal after closure by endogastric version.

Fbdy. 1120  1120	4 yrs.	Open safety-pin	Larynx, 2 days	None	Anterior con- firmation sure of larynx and gloscope	Open safe- ty-pin; avoidance of trauma and pre- vention of loss down- ward	Tucker alligator	Pointed branch	Extraction Cure 3 min.	Oral	Removed by point- ing method alligator form of Tucker forceps.
Fbdy. 1113  1113	8 mos.	Open safety-pin	Esophagus, 1 day	None	Laryn- goscope	Avoidance of trauma; open safe- ty-pin, point it ap- ward in an infant	Tucker	Pointed branch	Extraction Cure 55 sec.	Oral	Point-absorbed method of removal with Tucker fur- ceps.
Fbdy. 1122  1122	6 wks.	Open safety-pin	Esophagus, spring end below crico- pharyngeus, keep- er above, 1 day	None	Laryn- goscope	Open safe- ty-pin; avoidance of trauma, infant weighing 7 lbs.	Tucker alligator	Pointed branch	Extraction Cure 1 min.	Oral	Removed by point- ing method alligator form of Tucker forceps.
Fbdy. 1134  1134	9 mos.	Open safety-pin	Stomach, 5 days	None	6 mm. laryn- tro- scope	Open safe- ty-pin in stomach of infant	Rela- tion	Middle of pointed branch, to close, then spring end	Extraction Cure 26 min. 7 sec.	Oral	Blood found in stomach as soon as tube mouth en- tered. Pin found near pyloric end of stomach. Moved over to cardiac end and released by pulling back into tube mouth. Pin then released from forceps and regrasped by spring end and brought out closed.

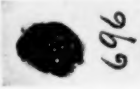


## SAFETY PINS—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1135 	6 yrs.	Open safety-pin	Esophagus below cricopharynx, 1 day	None	Anterior com- mis- sure laryn- go- scope	Open safety-pin; point up; avoidance of trauma	Alligator	Spring end	Extraction Cure	2 min. 59 sec.	Oral	Point-sheathed method.
Fbdy. 1140 	11 mos.	Open safety-pin	Esophagus and hypopharynx, 1 day	None	Laryn- goscope	Point em- bedded in right lat- eral wall	Rotation	Keeper	Extraction Cure	2 min. 5 sec.	Oral	Point-sheathed method.
Fbdy. 1138 	10 mos.	Open safety-pin	Right bronchus deep down below orifice of middle lobe, 13 days	None	4 mm.	Open safety-pin; point up, in tiny branch of infant	Side- curved	Keeper	Extraction Cure	10 min. 57 sec.	Oral	Subglottic and tracheal mucosal swelling. Pin withdrawn through tube after forceps manipula- tion against lip of bron- choscope tube mouth.
Fbdy. 1142 	11 wks.	Open safety-pin	Hypopharynx, 2 days	None	Laryn- goscope	Avoidance of trauma; 11 weeks' old infant	Rotation	Near point	Extraction Cure	2 min. 5 sec.	Oral	Version method.

# SEEDS, NUTS AND SHELLS





Fbdy. 633 	4 yrs.	Maize	Right bronchus at level of upper lobe bronchus, 24 days	None	5 mm. Bronchoscopic scope	For- pa spaces anal and in inspiration	Peanut	Extraction Cure	3 min. 4 sec.	Oral	Laryngotracheitis. Trachea full of pus. Grain of corn wedged tightly in bronchus.
Fbdy. 641 	6 yrs.	Orange seed	Strictured esophagus	None	7 mm.	Tight impaction; slippery surface	Side-curved	Extraction Cure		Oral	Removed from lumen of stricture. Tightly impacted.
Fbdy. 647 	4 yrs.	Peanut kernel	Right lower lobe bronchus, 25 days	None	5 mm.	None	Peanut	Extraction Cure	5 min. 26 sec.	Oral	Diffuse, laryngo-tracheo-bronchitis (bilateral). Pus discharge diffuse and profuse.
Fbdy. 658 	4 yrs.	Maize	Left main bronchus 6 days	None	5 mm.	Friable fleshy. Greatly swollen. Tight impaction	Side-curved	Extraction Cure	3 min. 8 sec.	Oral	Thick gummy pus in trachea. Coin tightly impacted in left bronchial orifice.
Fbdy. 682 	5 yrs.	Peanut kernel	Left main bronchus at orifice of upper lobe bronchus, 29 days	None	6 mm.	Friable, swollen, mucosa, pus	Peanut	Extraction Cure	5 min. 58 sec.	Oral	Larynx very much swollen. Pus coming out from nostrils. Trachea and bronchi inflamed. Rings invulnerable. Large quantity of pus coming up from right main bronchus. Mucosa of left bronchus more intensely red; more swollen.

## SEEDS, NUTS AND SHELLS—Continued


Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 696  696	2 yrs.	Peanut kernel	Right bronchus at orifice of upper lobe bronchus, 9 days	None	4 mm. Broncho- scope	Friable fbdy.	Peanut forceps	Minor axis	Extraction Cure	4 min. 20 sec.	Oral	Very firm stenosis just below carina. Diffuse tracheobronchitis with intensely red secretions thick and tough.
Fbdy. 719  719	3 yrs.	Watermelon seed	Left bronchus	None	5 mm.	None			Foreign body coughed up and swallowed. Recovered in stools	5 min. 49 sec.		Laryngotracheitis. Intense bronchitis right. Rather abundant mucopus. In- flamed, thick. Quite abundant mucopus. No foreign body found at bronchoscopy because previously coughed up.
Fbdy. 722  722	10 yrs.	Prune seed	Trachea, 1 day	None	6 mm.	Hard, slippery, shining surface	Side-curved	Minor axis	Extraction Cure	2 min. 20 sec.	Oral	Seed jammed at bifurcation with lower end in right bronchus. Bronchitis diffuse as far as could be seen. Exceedingly large fbdy. considering size of trachea. Remarkable child did not asphyxiate from jamming of fbdy. Pressure in trachea or subglottic region.

Fbdy. 723  723	16 yrs.	Cocklebur	Right lower lobe, 5 months		7 mm. Aspir- ing bron- cho- scope				Fbdy. not found at broncho- scopy. Coughed up after broncho- scopy. Recovery	35 min. 1 sec.		All interbronchial spurs lower lobe very much thickened. Mucosa erod- ed. Covered with granu- lations in spots. Middle lobe not bronchiectatic. Lower lobe bronchi mark- edly so. Most of pus seems to come from the lower lobe. The thickened inner surface of the inter- bronchial spurs makes some of the orifices seem stenosed. Below this apparent sten- osis the bronchi enlarge.
Fbdy. 724  724	2 yrs. 3 mos	Watermelon seed	Left main bronchus 6 days	None	5 mm.	None	Sid- e- curved	Flatwise	Fbdy. stripped off in mouth on with- drawal. Recov- ered in stools	5 min. 59 sec.		Foreign body at orifice of left main bronchus.
Fbdy. 725  725	21 mos	Peanut kernel	Right bronchus. Aspirated into left bronchus dur- ing bronchoscopy, from which it was removed. 2 weeks	None	5 mm.	Swollen mucosa. No for- eign spaces, until ob- tained by manipula- tion	Peanut	Minor axis	Extraction Cure	13 min. 33 sec.	Oral	Pus coming up when pa- tient coughed. Intense hyperemia. Large sub- mucosal ulceration in right bronchus. No for- eign body visible. All right lower lobe and mid- dle lobe bronchi intensely inflamed. Pus coming up from all branches. Pea- nut kernel found in left bronchus at orifice of up- per lobe bronchus. The ulceration in the right bronchus and the extreme hyperemia were cleared immediately after the pa- tient coughed up large quantities of pus render- ing it probable that the pres- sure of the cough on the right lung squeezed up the pus from the inflamed lower lobe and forced it into the trachea whence it was aspirated into the left lung.



## SEEDS, NUTS AND SHELLS—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 727  727	1 yr.	Peanut kernel	Right bronchus, 9 days	None	4 mm.	Friable object, tightly impacted in a swollen bronchus	Plain		Extraction Cure	3 min. 55 sec.	Oral	Intense laryngitis. Right bronchus swollen almost solid. Peanut kernel slightly impacted in swollen bronchus. Swollen mucosa. Not freely movable.
Fbdy. 732  732	4 yrs.	Watermelon seed	Right main bronchus, 4 days	None	5 mm.	None	Side- curved	Flatwise	Extraction Cure	2 min. 22 sec.	Oral	Intense diffuse tracheo- bronchitis.
Fbdy. 733  735	7 yrs.	Peanut ker- nel, 2 pieces	Both bronchi, 15 days	None	6 mm.	Friability of foreign bodies. Fragments of peanut kernel so large that removal possible to remove them through tube	Peanut	Minor axis	Extraction Cure	7 min.	Oral	Intense tracheobronchitis, diffuse in character. One portion of peanut kernel seen in left main bron- chus and two seen in right main bronchus.
Fbdy. 736  736	8 yrs.	Almond kernel	Right main bronchus, 9 days	None	6 mm.	None	Peanut		Extraction Cure	9 min. 30 sec.	Oral	Foreign body stripped off at glottis. Some frag- ments removed; others swallowed. Both bronchi clear on bronchoscopic inspection.




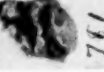

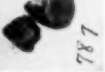
<p>Fbody. 738</p> 	<p>7 yrs.</p>	<p>Bean</p>	<p>Right bronchus, 15 days</p>	<p>None</p>	<p>6 mm.</p>	<p>Tight im- paction. Annular stricture almost against right bronchus rendered difficult the app- cation of forceps. It was nature of foreign body re- quired delicate grasp sufficient to with- draw it from its bed yet insuffi- cient to crush it</p>	<p>Peanut</p>	<p>Minor axis</p>	<p>Extraction 2 min. Cure 40 sec.</p>	<p>Oral</p>	<p>Intense laryngotracheitis. Subglottic edema very marked. Both main bronchial orifices equally swollen and red. Bean found occluding right bronchus at orifice of upper lobe bronchus. Bean grasped with delicate forceps and respirated portion went into left bronchus from which it was removed.</p>
---	---------------	-------------	------------------------------------	-------------	--------------	--	---------------	-------------------	---	-------------	---





## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- eign	Point of Seizure	Result	Time	Route	Comment
Fbdy. 743  743	2 yrs.	Chestnut kernel	Right bronchus, 1 week	None	5 mm.	Soft, fri- able, blood- stained pus over for- eign body	Peanut		Extraction Cure	7 min.	Oral	Left bronchus intensely in- flamed. Mucous membrane covered with tightly adherent patches of exu- date especially about the orifice of the upper lobe bronchus. Left lower lobe bronchus intensely inflamed. No foreign body visible. It was noted that the child be- came quite cyanotic as soon as a swab was intro- duced into the broncho- scope when the broncho- scope was in the left bronchus. On introduc- ing bronchoscope into right bronchus chestnut kernel was found in stem bronchus just below the upper lobe bronchus, tightly impacted. Right bronchus inflamed but not nearly so much as left. No exudate or ero- sion visible in right bronchus. It is quite evi- dent that the foreign body was, until within a few minutes or hours, located in left bronchus, having been coughed up and reaspirated into the right side.
Fbdy. 750  750	2½ yrs.	Peanut kernel	Right stem bronchus, 1 mo.	None	5 mm.	Soft, fri- able, al- limentary	Peanut		Extraction Cure	3 min. 18 sec.	Oral	Mild degree of bronchitis. Diffuse, very dry. Kernel found in right stem bronchus just below orifice of upper lobe bronchus. Free in lumen. Not clamped in swollen mucosa. Intense diffuse bronchitis, with copious mucous. Peanut kernel found in lumen of the bronchi was notice- ably absent in this case.



Fbdy. 756  756	8 yrs.	Peanut kernel	Trachea, 1 day	None	6 mm.	None	Peanut	Extraction Cure	5 min. 46 sec.	Oral	Subglottic tissues very much swollen. Larynx and trachea intensely inflamed. Pus in trachea equally inflamed. Foreign body lying back and forth between right bronchus and upper trachea being forced against the distal end of the bronchus, causing cough. Secretions abundant and blood-stained but not purulent so far as noticed.
Fbdy. 763  .763	15 mos.	Walnut kernel	Right main bronchus, 7 days	None	5 mm.	None	Forward grasping	Extraction Cure	7 min. 3 sec.	Oral	Larynx intensely swollen. Firm swelling of trachea which required considerable manipulation to pass walnut kernel completely occluding the right main bronchus at the level of the upper lobe bronchus. Firm swelling surrounding foreign body. Orifice of left bronchus swollen. Quantities of pinkish thick gummy pus removed by sponge pumping.
Fbdy. 767  767	3 yrs.	Peanut kernel	Left lower lobe bronchus, 4 weeks	None	5 mm.	No forceps spaces	Peanut	Extraction Cure	2 min. 24 sec.	Oral	Trachea and both bronchial orifices much swollen. Left main bronchus swollen nearly shut. Bronchial tags and all bronchial landmarks obliterated by the swollen mucosa. Gush of pus when peanut was removed. Gross appearances of pus were typically those of tracheobronchitis. The mucus was thick though not creamy; stringy, not "short." It was pinkish in color and its coming up copiously after removal of the peanut indicated a large area of "drowned lung," and the obstructive foreign body.



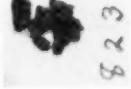
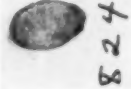
## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 769  769	6 yrs.	Peanut kernel	Trachea, 3 days	None	6 mm.	Great quantities of pink pus	Peanut	Minor axis	Extraction Cure	1 min. 6 sec.	Oral	Subglottic swelling very marked. High degree of tracheitis with consid- erable quantities of mu- coid-looking secretion.
Fbdy. 781  781	3 yrs.	Peanut kernel	Right main bron- chus, one portion at orifice of upper lobe bronchus, second portion in stem bronchus just below, 1 week	None	5 mm.	Very fria- ble; little cohesion; roasted	Peanut		Extraction Cure	3 min. 26 sec.	Oral	When first portion of pea- nut kernel was with- drawn the other was caught in the tube from which it was re- moved by reinsertion of bronchoscope.
Fbdy. 783  783	2½ yrs.	Maize	Moving between trachea and bron- chi; removed from left inferior lobe bronchus, 4 days	None	5 mm.	Seizure well be- yond the "germ" of the kernel	Plain	Beyond germ which was uppermost	Extraction Cure	4 min. 53 sec.	Oral	Diffuse tracheobronchitis. Patch of exudate about one square cm. in area at the orifice of the right main bronchus.
Fbdy. 787  787	21 mos.	Peanut kernel	One portion in trachea, other portion in left bronchus, 17 days	None	5 mm.	Soft, friable; pus	Peanut		One portion extracted Others coughed out through tube	1 min. 17 sec.	Oral	Trachea intensely red. No rings visible. Mucosa swollen.


Fbdy. 789  789	2 yrs.	Peanut kernel	Right bronchus at orifice of upper lobe bronchus, 19 days	None	5 mm.	None	None	Peanut	Minor axis	Extraction Cure	57 sec.	Oral	Entire half of peanut kernel found. Not tightly impacted.
Fbdy. 790  790	5 yrs.	Maize	Trachea, germ end up, 6 days	None	4 mm.	None	Beyond germ	Plain		Extraction Cure		Oral	Subglottic masses of swollen mucosa extending from both sides toward the center line making the subglottic lumen less than half of that of the glottis. Baby size tube used because of swollen condition of subglottis. Grain of corn found free in trachea slapping up against tube mouth twice before it was seized and removed.
Fbdy. 792  792	8 yrs.	Prune seed	Trachea, 3 days	None	6 mm.	None	Minor axis	Plain cupped centers		Extraction Cure	4 min. 49 sec.	Oral	Extremely large also and very plump shape of prune seed accounted for extreme dyspnea of child on admission. Relieved great the moment prune seed was out.
Fbdy. 799  799	3 yrs.	Peanut kernel	Right main bronchus near orifice of middle lobe bronchus, 16 days	None	5 mm.	Extremely swollen condition of the bronchial mucosa		Plain		Extraction Cure	7 min. 4 sec.	Oral	Severe diffuse laryngo-tracheo-bronchitis. Marked subglottic swelling. Displaced subglottic space from middle to upper lobe bronchus full of mucopurulent secretion. Peanut kernel grasped and withdrawn but broke on emerging from glottis. One fragment wedged in trachea and was found wedged in orifice of left upper lobe bronchus.

## SEEDS, NUTS AND SHELLS—Continued

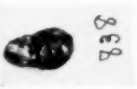



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 800 	14 mos.	Portion peanut shell	Trachea subglottic, about 2 hours	None	4 mm.	None	Alligator	Flatwise	Extraction Cure	30 sec.	Oral	Piece of peanut shell completely sagittally in trachea, immediately below glottis.
Fbdy. 802 	2 yrs. 11 mos.	Peanut kernel	Right bronchus just below bifurcation, 11 days	None	5 mm.	No forceps spaces	Peanut		Extraction Cure	13 min. 2 sec.	Oral	Small fragment of peanut kernel tightly impacted in swollen right bronchus just below bifurcation. Inhaled while child was shut. Subglottic trachea greatly swollen, causing acute stenosis just below larynx.
Fbdy. 805 	3 yrs.	Maize	Trachea, 6 days	None	5 mm.	None	Peanut	Flatwise beyond germ	Extraction Cure	2 min. 24 sec.	Oral	Very mild degree of tracheo-bronchitis.
Fbdy. 815 	2 1/2 yrs.	Peanut kernel	Left main bronchus, 4 days	None	5 mm.	No forceps spaces	Peanut		Extraction Death 3 days after discharged well, from apparently "croup" (subglottic edema?)	1 min. 12 sec.	Oral	Marked subglottic swelling. Pinkish very tenacious, mucopurulent secretion covered mucosa; not very copious in amount. Left main bronchus full of secretion up to the level of the carina. No straining through secretion.

Fbdy. 819 	4 yrs.	Watermelon seed	Left main bronchus 6 days	None	5 mm.	None	Plain	Flatwise	Extraction Cure	10 min.	Oral	Thick creamy pus very tenacious, adhering to trachea and both main bronchi. Fbdy. heard beating against glottis just before it was removed. It was introduced, but it must have been quickly aspirated into the left bronchus where it was found with bronchoscope.
Fbdy. 822 	5 yrs.	Maize	Trachea, 5 days	None	5 mm.	None	Sid- curved	Flatwise beyond form	Extraction Cure	1 min. 52 sec.	Oral	Exudate attached to the subglottic mucosa. Removal of one side left a blood-stained, leukodiphtheritic membrane.
Fbdy. 823 	2 yrs.	Peanut kernel	Trachea, 7 days	None	5 mm.	None	Peanut		Extraction Cure	9 min. 48 sec.	Oral	Diffuse tracheobronchitis. Mucoid subglottic alluring rings of trachea invisible.
Fbdy. 824 	5 yrs.	Watermelon seed	Left main bronchus, 16 days	None	5 mm.	None	Plain	Flatwise	Extraction Cure	1 min. 12 sec.	Oral	Foreign body found with its largest plane exposed. Absorbed by both the inner and outer sides of the watermelon seed.

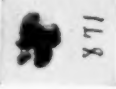
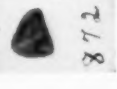
## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 827  827	2 yrs.	Watermelon seed	Trachea, 5 days	None	5 mm.	None	Side-curved	Flatwise	Extraction Cure	5 min. 40 sec.	Oral	
Fbdy. 828 No Specimen	3 yrs.	Peanut kernel	Right main bronchus, 12 days	None	5 mm.	None	Peanut		Extraction Cure	6 min. 3 sec.	Oral	Right main bronchus obstructed by pultaceous masses of peanut kernel. When removed it came away in small fragments in secretions. Diffuse tracheobronchitis less marked on left side; very intense on right side especially at location of peanut kernel and also in the subglottic region.
Fbdy. 832  832	2 yrs. 8 mos.	Chestnut kernel	Left main bronchus, 10 days	None	5 mm.	None	Peanut		Portion removed; remainder coughed out through tube; cure	1 min. 3 sec.	Oral	Two granulation buds seen where nut kernel had lain.
Fbdy. 833  833	2 yrs.	Coffee berry fragments	Left lower lobe bronchus, 2 weeks	None	5 mm.	Tight impaction in bronchus	Side-curved		Extraction Death	20 min.	Oral	Death from lobar pneumonia 1 month after removal of foreign body.



Fbdy. 838 	1 yr.	Maize	Left main bronchus, 3 days	None	4 mm.	Inflammatory acute abscess of bronchus	Plain		Extraction Cure	1 min. 2 sec.	Oral	Subglottic edema; diffuse laryngotracheobronchitis; left bronchus dilated and swollen almost shut; erosion with reddened edges almost all way around the left bronchial orifice. Edematous orifice dilated with forceps and grain of corn removed, leaving off of the left upper lobe bronchus.
Fbdy. 839 	22 mos.	Watermelon seed	Right main bronchus at level of middle, about 3 months	None	5 mm.	Flooding of pus	Plain	Flatwise	Extraction Cure	1 min. 3 sec.	Oral	Large amount of pus in trachea and coming up both bronchi.
Fbdy. 846 	23 mos.	Peanut kernel	Right main bronchus, 19 days	None	5 mm.	Acute inflammatory abscess of bronchus	Plain		Extraction Death	5 min. 23 sec.	Oral	Larynx somewhat inflammatory above cords; subglottic redness, swollen, inflamed and swollen. Trachea in high state of inflammation. Right bronchial orifice swollen and very red. Left bronchial orifice much less so. Abscess right bronchus drained bronchoscopically 13 days after removal of body. Death a week later from rupture of lung abscess and liberation of a large accumulation of pus.
Fbdy. 856 	21 mos.	Peanut kernel	Occluding orifice of right upper lobe bronchus, 3 mos.	None	4 mm.	Location of body in upper lobe bronchus. Swollen, inflamed, redness and slippery-ness of body.	Peanut		One portion removed, other portion collapsed out thro' bronchoscope	1 min. 32 sec.	Oral	Diffuse tracheobronchitis; not nearly so much secretion as usually seen in peanut cases.

## SEEDS, NUTS AND SHELLS—Continued

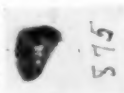
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 871 	2 yrs.	Peanut kernel fragments	Left main bronchus, at orifice of upper lobe, about 3 months	None	5 mm.	Great quantities of pus	Peanut		Two portions retained; no more; no coughed out through bronchoscope	3 min. 39 sec.	Oral	Trachea chronically inflamed; no rings visible. Left bronchus completely full of pus (full of pus, but no air passing. No cough could expel it until after the swab had been introduced two or three times. Left main bronchus elliptical in outline; great swelling of inner wall; peanut kernel immediately below granulation tissue, very soft under forceps; large quantity of thick, creamy pinkish pus liberated by removal of peanut. Removal of peanut by suction and by forceps. Entire lower lobe seems to be involved in either drowned lung or abscess, probably partially both.
Fbdy. 872 	2 yrs.	Portion almond kernel	Right inferior lobe bronchus immediately below orifice of middle lobe bronchus, about 3 months	None	5 mm.		Peanut	Equator	Extraction Cure	2 min. 2 sec.	Oral	

Subglottic edema so great as to present considerable obstruction to the introduction of the 4 mm. bronchoscope. The right bronchus was completely obliterated by swelling of mucosa; upper portion of left main bronchus noted in passing was distinctly inflammatory though somewhat less so than right. In right bronchus very much swollen in its upper part; peanut kernel was moving up and down in respiratory current; at each inspiration it would go down and escape for only of brief passage for air on three of its sides; on expiration it would move upward and fit tightly in the edematous portion of the bronchus. The action noted was a "ball valve" with the ball rising from below and fitting tightly into the edematous portion of the bronchus which served as a valve-seat, the direction of the action usually noted in peanut cases; namely, the expansion of the bronchi at the site of the peanut kernel making ample by-passage spaces, which were immediately closed at the beginning of expiration.

Bronchial mucosa on right side very red and swollen; lumen diminished to not more than one-third average; orifice diameter of a child's finger. Peanut slipped off forepart of pharynx and was swallowed.

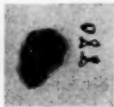
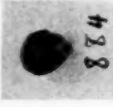


Fbdy. 875	2 yrs.	Peanut kernel	Right main bronchus below orifice of upper lobe of bronchus, 6 days	None	4 mm.	Acute inflammatory stenosis of bronchus	Peanut	Extraction Cure	1 min. 55 sec.	Oral	
Fbdy. 877 No Specimen	3 yrs.	Peanut kernel	Right inferior lobe of bronchus, 8 days	None	5 mm.	Acute inflammatory stenosis of bronchus	Moroquito	Extraction Cure	14 min. 59 sec.	Oral	

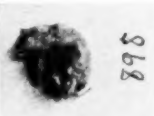
Fbdy. 875



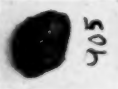
Fbdy. 877  
No Specimen





## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 883 	15 mos.	Peanut kernel	Right main bronchus, 9 days	None	4 mm.	Acute inflammation, edema, and consolidation of bronchus	Peanut		Extraction Cure	1 min. 5 sec.	Oral	Subglottic region swollen and inflamed; mucous membrane thick, tenacious pus in trachea and orifices of both bronchi.
Fbdy. 884 	31 mos.	Peanut kernel	Right main bronchus, 2 days	None	5 mm.	Large size and friability of body.	Peanut		Extraction Cure	7 min. 3 sec. 5 min. 56 sec.	Oral	Subglottic edema worse on left side. Laryngotracheobronchitis not so severe as in previous cases but the subglottic edema fully as marked as in the most severe cases. Second portion of peanut kernel removed bronchoscopically one week after removal of first portion.
Fbdy. 890 	4 yrs.	Peanut kernel	Right main bronchus, 7 days	None	5 mm.	Tight impaction and friability	Peanut		Extraction Cure	23 min. 47 sec.	Oral	
Fbdy. 891 	27 mos.	Peanut kernel	Trachea at bifurcation, obstructing both bronchi, 8 days	None	5 mm.	Threatened asphyxia	Peanut		Extraction Cure	2 min. 5 sec.	Oral	Marked swelling of the subglottic area and of the tracheal mucosa.

Fbdy. 892 	2 yrs.	Peanut kernel	Trachea, 2 days	None	5 mm.	Quantities of pus	Side-curved	Extraction Cure	30 sec.	Oral	Foreign body surrounded by a thick accumulation of pus.
Fbdy. 898 	15 yrs.	Portion corn stalk	Right stem bronchus, 5 weeks	None	6 mm.	No forceps seen even on inspiration	Side-curved	Extraction Cure	3 min. 25 sec.	Oral	All of L's bronchi on right side very much inflamed. Left bronchial orifice likewise. Deeper part of left bronchus not explored. After removal small bronchi noticed to be surrounding themselves at entrance of pus.
Fbdy. 900 	3 1/2 yrs.	Bean	Trachea, 2 days	None	5 mm.	Large size of foreign body jammed in glottic chink	Fence-trated	Extraction Cure		Oral	Tracheotomy required for urgent dyspnea; bean then found above tracheotomy cannula and removed orally.
Fbdy. 904 	5 1/2 yrs.	Peanut kernel	At junction of left upper lobe bronchus with stem bronchus	None	6 mm.	Forceps spaces on one side only	Fence-trated	Extraction Cure	3 min. 2 sec.	Oral	Subglottic swelling; acute laryngotracheobronchitis with a large quantity of mucus secreted and adherent to the mucosa everywhere. Left bronchial orifice more swollen than the right.

## SEEDS, NUTS AND SHELLS—Continued




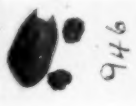
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 905 	3 yrs.	Peanut kernel	Right main bronchus at orifice of upper lobe, 1 week	None	5 mm.	Acute inflammatory stenosis, friable body.	Fenestrated		Extraction Cure	1 min. 59 sec.	Oral	Diffuse laryngotracheobronchitis. Secretions very thick and tough, but scanty in quantity.
Fbdy. 911 	11 mos.	Watermelon seed	Trachea, 4 days	None	4 mm.	None	Plain	Flatwise	Extraction Cure	4 min. 39 sec.	Oral	Intense subglottic swelling nearly meeting in the median line; intense tracheitis.
Fbdy. 918 	4 yrs.	Watermelon seed	Trachea, 3 days	None	5 mm.	Slippery moving body.	Side-curved	Flatwise	Extraction Cure	23 sec.	Oral	Larynx and trachea very edematous. Not a great deal of secretion present.

Fbdy. 910 	2 yrs.	Watermelon seed	Trachea, 3 days	None	5 mm.	Fbdy. moving about	Side-curved	Flatwise	Extraction Cure	1 min. 3 sec.	Oral	Watermelon seed free in trachea causing a fluttering, flapping sound as soon as the bronchoscope entered. Secretion very copious. Inflammation intense. Trachea swollen.
Fbdy. 920 	3 yrs.	Peanut kernel	Right main bronchus, 15 days	None	5 mm.		Fence-traited		Extraction Cure	1 min. 1 sec.	Oral	Diffuse laryngotracheobronchitis. Pharynx intensely inflamed.
Fbdy. 924 	17 yrs.	Grape seed	Esophagus	None	8 mm.	Moving about in folds	Plain cupped			1 min. 58 sec.	Oral	Removed during esophagoscopy for treatment of phrenospasm.
Fbdy. 927 	5 yrs.	Watermelon seed	Trachea, 5 days	None	5 mm.	Constantly changing position of forceps spaces	Side-curved	Flatwise	Extraction Cure	3 min. 34 sec.	Oral	Moderate degree of subglottic swelling. Marked laryngotracheitis with very scanty secretions.

## SEEDS, NUTS AND SHELLS—Continued

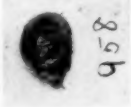


Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Saisure	Result	Time	Route	Comment
Fbdy. 928 	15 mos.	Watermelon seed	Trachea, 70 days	None	5 mm.	Constantly shifting forceps spaces	Slide-curved	Flatwise	Extraction Cure	2 min. 45 sec.	Oral	Laryngotracheitis is very marked. Subglottic swelling on both sides projecting toward the midline. Considerable quantity of tough secretion visible in trachea.
Fbdy. 931 	3½ yrs.	Maize	Trachea, 16 days	None	5 mm.	Forceps spaces very small	Alligator	Flatwise	Extraction Cure	26 sec.	Oral	Immediately below glottis.
Fbdy. 932 	58 yrs.	Peanut kernel	Right inferior lobe bronchus, 45 days	Local	7 mm.	Lodged in small branch bronchus	Plain cupped		Extraction Cure	13 min. 52 sec.	Oral	Mucosa of entire right bronchial tree so far as visible inflammatory. Superficial erosion at site of peanut kernel.
Fbdy. 936 	16 mos.	Peanut kernel	Right bronchus at orifice of upper lobe bronchus, 32 3/4 days	None	5 mm.	Acute inflammation only, friable object	Plain peanut		Extraction Cure	9 min. 35 sec.	Oral	







Fbdy. 939  939	22 mos.	Maize	Right main bronchus, 4 days	None	5 mm.	No forceps spaces	Side-curved	Flatwise	Extraction Cure	9 sec.	Oral	Secretions rather dry and scanty.
Fbdy. 940  940	18 mos.	Portion apple stem	Left lower lobe bronchus, about 3 months	None	5 mm.	Enormous amount of secretion	Mosquito		Foreign body expelled through bronchoscope	6 min. 15 sec.	Oral	Secretions abundant; tracheal mucosa highly inflammatory; left lower lobe bronchus; very inflamed; lumen collapsed anteroposteriorly during expiration; small dark object seen at orifice of left upper lobe bronchus; displaced with closed forceps; seemed free to move.
Fbdy. 944  944	3 yrs.	Watermelon seed	Trachea, 22 days	None	5 mm.	Shifting	Plain	Flatwise		5 min. 30 sec.	Oral	Shifting foreign body fixed with tube mouth for seizure.
Fbdy. 946  946	4 yrs.	Peanut kernel	Right bronchus at orifice of upper lobe bronchus, 16 days	None	5 mm.	Friable; mucosa swollen	Plain to dislodge; lemon-treated peanut	Minor axis	Extraction Cure	4 min. 2 sec.	Oral	Mucosa swollen.

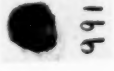



## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 948  948	6 yrs.	Maize	Right stem bronchus, 2 days	None	6 mm.	Large size of swollen fbody.	Plain	Flatwise beyond germ	Extraction Cure	5 min. 45 sec.	Oral	Intense laryngotracheo-bronchitis, subglottic edema; trachea inflamed; middle line; both main bronchial orifices swollen to about two-thirds of their normal diameter; grain of corn quite soft to grasp; tightness of fit in glottis prevented removal until third grasping.
Fbdy. 949  949	16 mos.	Maize	Trachea at bifurcation, 12 days	None	5 mm.	Threatened asphyxia	Side-curved	Flatwise beyond germ	Extraction Cure	1 min. 20 sec.	Oral	Supraglottic and sub-glottic trachea inflamed; subglottic tissue bulging toward the median line; tracheal wall swollen.
Fbdy. 953  953	15 mos.	Peanut kernel	Trachea, 13 days	None	4 mm.	Shifting; friable fbody.	Fenestrated	Minor axis	Extraction Cure	2 min. 4 sec.	Oral	Diffuse tracheobronchitis.
Fbdy. 956  956	3 yrs.	Jobs tear	At orifice of left inferior lobe bronchus, 14 days	None	5 mm.	Hard, smooth surfaced fbody, fitting tightly	Peanut for- ceps; then cupped.		Extraction Cure	7 min. 20 sec.	Oral	Dislodgement of fbody, liberated a large amount of pus; fbody, respiration into right side from which it was removed.



Fbdy. 958 	7 yrs.	Maize	Occluding left lower lobe bronchus, 10 days	None	6 mm.	No forceps space—4	Side-curved	Flatwise beyond germ	Extraction Cure	Oral	Grain of corn found entirely occluding the left lower lobe bronchus, partially occluding the left upper lobe bronchial orifice; grain being tightly wedged in the lower lobe bronchus for about two-thirds of its length. It was removed after rotating sagittally.
Fbdy. 962 	2 yrs.	Bean	Trachea obstructing both bronchi at times, about 13 hours	None	5 mm.	Frailility of foreign body; threatened asphyxia	Encased		Extraction Cure	2 portions removed orally, remaining in posterior portion withdrawn with 'pelled thro' tracheotomy; colonized by same pump-wound	Urgent dyspnea. Bean found lying across the carina, obstructing both bronchi. Tilting immediately relieved breathing. Fbdy. very friable and soft; broke in forceps; fragments were drawn through tube. One large portion withdrawn with bronchoscope deemed inadvisable. Large quantity of thick mucilaginous secretion was removed from trachea by suction pumping. Tracheotomy required next morning to prevent drowning of patient in his own secretion. Large amount of thick mucus expelled from cannula, relieving dyspnea. Remaining portion of foreign body expelled through tracheotomy wound.
Fbdy. 964 	22 mos.	Peanut kernel	Right stem bronchus, just below orifice of upper lobe, 3 days	None	5 mm.	Tight impaction	Slender, then peanut	Minor axis	Extraction Cure	Oral	Forceps spaces exceedingly small; slender forceps applied during deep inspiration and kernel withdrawn upward to trachea; seized with peanut forceps and drawn into oral cavity, being then swallowed by patient. Fbdy. recovered in stools.

## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 969  969	61 yrs.	Orange seed	In malignant stenosis of esophagus	None	9 mm.	Slippery surface of fbdy.	Plain cupped		Extraction	7 min. 9 sec.	Oral	Fbdy. found in a malignant stricture 20 cm. from upper incisor teeth.
Fbdy. 973  973	27 mos.	Almond kernel	In trachea and left lower lobe bronchus, 3 days (two different portions)	None	5 mm.	Fbdy. multiple; inflammatory stenosis of bronchi	Side-curved		Extraction	24 min. 4 sec.	Oral	Portion of nut kernel found in trachea and another portion tightly impacted in left lower lobe bronchus. Laryngotracheitis of high degree and bronchitis. Food got out both lungs in the left bronchus there are patches of exudate and a small amount of very tough secretion.
Fbdy. 976  976	20 mos.	Canna seed	Right stem bronchus, 42 days	None	5 mm.	Tight impaction; smooth, very hard fbdy.	Side-curved cupped face		Extraction Cure	1 min. 21 sec.	Oral	
Fbdy. 980  980	33 yrs.	Cocklebur	Larynx, 2 days	None	Anterior, transverse, insure laryngoscope	None	Alligator		Extraction Cure	32 sec.	Oral	Burr found imbedded in ulceration in the anterior portion of the larynx, removed by curettage. Reaction to dislomb it. No cough.




Fbdy. 991 	2 yrs.	Peanut kernel	Left lower lobe bronchus, 12 days	None	5 mm.	Acute inflammatory stenosis	Side-curved		Extraction Cure	8 min. 47 sec.	Oral	Subglottic tissue somewhat swollen; tracheal mucosa very much swollen, rendering rings invisible; left bronchus swollen almost shut, the remaining space being occluded by secretions which seemed to be mucopurulent rather than pure pus.
Fbdy. 1002 	1 1/2 yrs.	Peanut kernel	Trachea, 3 days	None	5 mm.	Acute inflammatory stenosis	Side-curved	Minor axis	Extraction Cure	2 min. 40 sec.	Oral	Intensely swollen subglottic tissue; intense tracheitis; secretions thick and tenacious.
Fbdy. 1004 	7 yrs.	Coffee berry	Left stem bronchus, 16 days	None	3 mm.	Acute inflammatory stenosis	Side-curved	Minor axis	Extraction Cure	5 min. 20 sec.	Oral	Left bronchus filled with pus which was called upon for getting this away before coffee berry could be seen. Mucous in stem bronchus below orifice of upper lobe bronchus; in a moment a fresh quantity of thick pus came up and occluded the orifice of the upper lobe bronchus, the orifice of which was swollen so that its diameter was not more than half of that of the normal in a child of this age.
1019 	10 yrs.	Peanut kernel	Right stem bronchus below orifice of middle lobe bronchus, 3 days	None	6 mm.	Swollen; aspirating foreign body	Side-curved	Over equator	Extraction Cure	4 min. 25 sec.	Oral	Stem bronchus above middle lobe obstructed by mass of granular swollen mucosa of which the largest mass was projected anteriorward from anterior wall.

## SEEDS, NUTS AND SHELLS—Continued

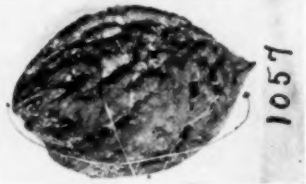

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
No. 1024 No specimen	27 mos.	Peanut kernel (?)	Orifice of left lower lobe bronchus, 8 days	None	4 mm. aspir- ating	Swollen mucosa; friable foreign body	Side- curved		Extraction Cure	7 min. 13 sec.	Oral	Left bronchus swollen and closing completely on in- spiration; plug of pus in orifice of left lower lobe bronchus; felt firm to touch and adherent; tube contained peanut kernel. Withdrawn through glot- tis and lost in pharynx.
Fbdy. 1044 	5 1/2 yrs.	Peanut kernel	Right stem bron- chus, 3 weeks	None	5 mm. aspir- ating	Swollen mucosa; friable foreign body	Side- curved	Minor axis	Extraction Cure	6 min. 7 sec.	Oral	
Fbdy. 1108 	4 yrs.	Peanut kernel	Right bronchus at orifice of upper lobe, 13 days	None	5 mm. aspir- ating	Swollen mucosa; friable foreign body	Side- curved		Extraction Cure	2 min. 10 sec.	Oral	Bronchial mucosa swollen; moderate amount of pus present.

Fbody, 1066:	22 mos.	Peanut kernel	Left main bronchus between bifurcation and orifice of upper lobe, 1 day	None	5 mm.	Swollen mucosa; friable foreign body	Slightly curved	Equator	Extraction	Death	2 min.	Oral	Subglottic tissues somewhat swollen. Trachea and both bronchial orifices intensely red and inflammatory. Second bronchoscopy: larynx swollen above and below. Trachea swollen and compressed from posterolateral wall. Carina unusually broad. Right main bronchus swollen entirely about middle third of segment. No foreign body seen. Bronchus dilated and secretions aspirated.
Fbody, 1031	22 yrs.	Grain of corn (fresh)	In strictured esophagus, 4 days	None	7 mm.	Foreign body tightly wedged in strictured esophagus like cork in bottle	Plain	Flatwise, advancing forceps well on top of center of foreign body	Extraction	Recovery	3 min.	Oral	After removal of corn stricture seen to be about 3 or 4 mm. in diameter at top. About 1 cm. deeper down was seen a smaller stricture and below that point the lumen of stricture deviated.
Fbody, 1072	4 yrs.	Grain of corn (maize)	Right main bronchus, 2 days	None	5 mm. aspirating	Shifting forceps spaces		Flatwise	Extraction	Cure	2 min. 52 sec.	Oral	Grain of corn found loose in trachea; on deep inspiration aspirated to right bronchial orifice. Violent diffuse tracheobronchitis; subglottic tissue much swollen.
Fbody, 1079	5 yrs.	Grain of corn (maize)	Right main bronchus, 11 days	None	5 mm. aspirating	Swollen kernel, tightly impacted; no spaces small and only on inspiration	Light side-curved		Extraction	Cure	15 min.	Oral	Bronchoscope filled with mucopurulent secretion on insertion. Corn engaged end of tube and immediately aspirated. Mucosa of trachea and bronchi very inflammatory and bathed in mucus. Lumen of trachea and bronchi completely closed on rough inspiration when to double original size.



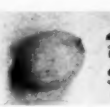
## SEEDS, NUTS AND SHELLS—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Suture	Result	Time	Route	Comment
Fbdy. 1127 	14 mos.	Grain of corn (maize)	Right main bronchus, few hours	None	4 mm. aspirating	None	Side-curved	Flatwise, beyond edge	Extraction Cure	2 min. 14 sec.	Oral	Marked swelling of larynx and subglottic tissue. Trachea and bronchus inflamed. Whitish secretion present. Foreign body found loose in trachea, but was aspirated into right bronchus during inspiration. Tracheotomy necessary because of subglottic edema.
Fbdy. 1144 	3 yrs.	Grain of corn (maize)	Trachea, 9 days	None	5 mm.	Shifting forceps spaces	Side grasping	Flatwise beyond edge	Extraction Cure	3 min. 10 sec.	Oral	Grain of corn loose in trachea. Considerable thin secretion present.
Fbdy. 1042 	11 yrs.	Portion almond shell	Right stem bronchus, 3 months	None	6 mm. aspirating	Foreign body in granulating abscess	Side-curved	Flatwise	Extraction Recovery	8 min. 5 sec.	Oral	On insertion of bronchoscope very foul stale odor quite characteristic of prolonged sojourn of foreign body came through bronchus. Trachea and left bronchus inflamed. Right bronchus lined with granulation tissue bleeding freely. Large quantity of pus liberated as soon as almond shell removed.



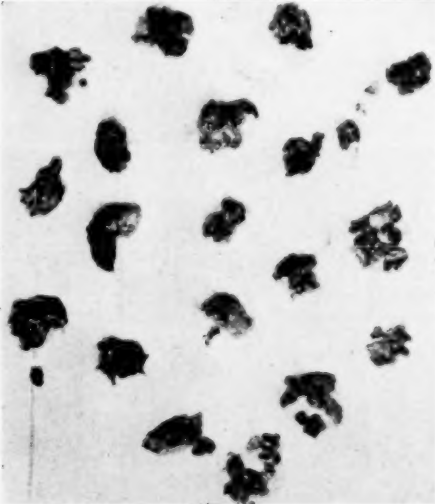

<p>Body. 1057</p> 	<p>33 yrs.</p>	<p>Pearl stone</p>	<p>Esophagus below cricopharyngeal fold, 2 days</p>	<p>None</p>	<p>Short, lippled specu- lum Forbes</p>	<p>Tight im- paction; large size</p>	<p>Alligator rota- tion</p>	<p>Minor axis</p>	<p>Extraction Cure</p>	<p>13 min. 39 sec.</p>	<p>Oral</p>	<p>Larynx and subglottic structures red and edematous, bathed with thin, white mucus. Bronchoscope met with slight resistance at the glottis. Bean was coughed into end of tube and immediately reaspirated into right main bronchus, the upper end of tube being above the carina. Bean grasped with side curved forceps over minor axis, withdrawn against bronchoscope. Bronchoscope forceps and bean withdrawn. Bean brought into glottis where it met with firm resistance and when steady traction was made bean broke in middle, lower half remaining in trachea, upper half in right main portion of hull removed with bronchoscope. Bronchoscope reinserted. Trachea contained whitish pus which was aspirated and remaining lower half of bean was coughed and reaspirated into right main bronchus. Remainder of hull coughed out through bronchoscope. Remaining portion of bean was in two pieces, smaller portion removed through bronchoscope; larger was brought against end of tube and removed along with bronchoscope.</p>
<p>Body. 1068</p> 	<p>5 yrs.</p>	<p>Bean</p>	<p>Right main bronchus, 3 days</p>	<p>None</p>	<p>6 mm.</p>	<p>Soft, pulpy; macerated friable foreign body with loose cortex</p>	<p>Side- curved</p>		<p>Extraction Cure</p>	<p>12 min. 30 sec.</p>	<p>Oral</p>	


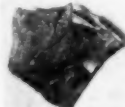
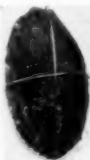

## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1043 	2 yrs.	Watermelon seed	Trachea, 8 days	None	5 mm.	Friable foreign body	Plain	Flatwise	Extraction Cure	2 min. 10 sec.	Oral	
Fbdy. 1048 	5 yrs.	Watermelon seed	Right bronchus at orifice of upper lobe bronchus, 5 days	None	5 mm. aspirating	Tight impaction; swollen mucosa	Side-curved	Flatwise	Extraction Cure	1 min. 7 sec.	Oral	Bronchial mucosa very inflammatory, thick and velvety.
No. 1090 No Specimen	2 yrs. 4 mos.	Watermelon seed (?)	Left bronchus, 1 month	None	5 mm.	Swollen mucosa	Side-curved	Flatwise	Extraction Cure	13 min. 50 sec.	Oral	Left bronchus completely closed on expiration by polypoid granulations protruding from wall. Small firm mass of unknown character grasped with forceps. Part removed in forceps and part stripped off in pharynx.
Fbdy. 1020 	5 yrs.	Job's tear (seed of lachrima Jobi)	Right bronchus at orifice of upper lobe bronchus, 1 day	None	5 mm. aspirating	Smooth conical foreign body, impacted	Plain	Minor axis	Extraction Cure	6 min. 50 sec.	Oral	Bead loose in trachea, on inspiration it would go into right bronchus. Removed from this location.


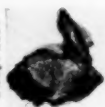

<p>Fbdy. 1021</p> 	2 yrs.	Cedar leaves	Pharynx overhanging orifice of larynx, 5 hours	None	None	Prevention of dislodgment by downward pressure	Angular with tongue depressor		Extraction Cure		Oral	Spines anchored in mucosa.
<p>Fbdy. 1030</p> 	43 yrs.	Egg-shell	Esophagus in middle third	None	9 mm.	Friable foreign body imbedded in ulcer	Plain	Flatwise	Extraction Cure	11 min. 25 sec.	Oral	Esophagus found swollen shut; intensely inflamed and an ulcerated bleeding point found on posterior wall with egg-shell imbedded in ulceration.
<p>No. 1098 No Specimen</p>	20 mos.	Peanut kernel	Left bronchus, 5 days	None	5 mm.	Swollen; shreds of membrane	Gauze sponges		Recovery	3 min. 20 sec.	Oral	Right bronchus normal. Left bronchial orifice nearly closed; mucosa swollen, red and edematous. Shreds of membrane removed with thick crusts. No peanut kernel found but a number of tiny crumbs seen attached to mucosa.
<p>Fbdy. 1118</p> 	2 yrs.	Peanut kernel	Orifice of right bronchus, 1 day	None	5 mm.	Swollen mucosa; friable foreign body	Side-curved	Equator	Extraction Cure	1 min. 25 sec.	Oral	Subglottic tissue very much swollen, considerable amount of pus in trachea and bronchi.



## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Suture	Result	Time	Route	Comment
Fbdy. 1119	2 yrs.	Bronchial casts (18 casts and crustinall)		None	5 mm.	Respiratory arrest; death impending; no obstruction of both main bronchi	Side-curved		Recovery			Same case as above. Second bronchoscopy showed inflamed bronchi and casts with thick crustified secretion. Particles of which were removed with forceps. After withdrawal of bronchoscope child coughed up a crust representing a partial cast of a bronchus. Bronchoscopy repeated five days; each time impending death by asphyxia was averted.
Fbdy. 1123 	2 yrs.	Peanut kernel	At orifice of right bronchus; portion projecting into trachea, 3 days	None	5 mm.	Friable foreign body	Side-curved	Minor axis	Extraction Cure	57 sec.	Oral	





Fbdy. 1145  1145	13 mos.	Peanut kernel	Left main bronchus, 2 days	None	4 mm.	Friable foreign body	Slide-curved	Minor axis	Extraction Cure	7 min. 45 sec.	Oral	Slight swelling of subglottic tissues. Foreign body bathed in thin whitish pus. Peanut in left main bronchus just beyond carina. Kernel coughed up freely during removal; portion of nut remaining in trachea. Pus came up freely from below nut kernel.
Fbdy. 1124  1124	42 yrs.	Portion crab shell	Esophagus just below cricopharyngeal muscle, 5 days	None	Forbes speculum	Proper presentation of folds	Rotation	Flatwise	Extraction Cure	1 min. 24 sec.	Oral	
Fbdy. 1126  1126	3 yrs.	Prune seed	Esophagus, 2 days	None	7 mm.	Hard	Plain	Minor axis	Extraction Cure	1 min. 15 sec.	Oral	Old lye stricture case. Foreign body found embedded in strictured lumen of esophagus. Zone of esophagitis found surrounding location of foreign body.
Fbdy. 1139  1139	13 mos.	Bean	Right main bronchus extending above carina, 1 day	None	4 mm.	Soft, pulpy friable foreign body	Side grasping	Equator	Extraction Cure	16 min.	Oral	Marked swelling of subglottic tissues and tracheal mucosa. Whitish secretion present in trachea.

## SEEDS, NUTS AND SHELLS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1141  1141	2 yrs.	Walnut kernel	Right stem bronchus, 4 days	None	5 mm.	Friable foreign body	Side grasping	Equator	Extraction Cure	5 min. 46 sec.	Oral	Small amount of thick secretion found in trachea which was aspirated. Mucous membrane of trachea inflamed. Pus coming from right bronchus.
Fbdy. 1133  1133	19 yrs.	Portion of cork	Right bronchus below orifice of middle lobe, 1 month	Local	8 mm. aspirating	Foreign body below stenosis	Side-curved	Equator	Extraction Cure	1 min. 47 sec.	Oral	Projecting masses of granulations lining right bronchus from the orifice of upper lobe bronchus downward; cork found below a point completely occluded by granulation tissue.
Fbdy. 1143 	39 yrs.	Portion of clothing (shirt)	Left lower lobe bronchus, 2 years	None	Pleuroscope	Finding foreign body in fistulae	Side-curved		Extraction		Thru wound in chest; pleuroscopy	Foreign body removed during retrograde bronchoscopy. There was no air in left chest explored. There was marked pulsatory movement of lung. A large bronchus with the lumen almost completely plugged could be made into a large plug about $\frac{3}{4} \times 1\frac{1}{2}$ inches in diameter and full lumen of bronchus revealed. Considerable pus was present and air bubbled through freely.

<p>Fbdy. 1152</p>  <p>1152</p>	<p>18 mos</p>	<p>Grain of corn (maize)</p>	<p>Right bronchus below orifice of upper lobe bronchus, 12 days</p>	<p>None</p>	<p>5 mm.</p>	<p>Swollen mucosa of foreign body tightly impacted; no deep spaces</p>	<p>Side-curved</p>	<p>Flatwise beyond edge</p>	<p>Extraction Cure</p>	<p>3 min.</p>	<p>Oral</p>	<p>Diffuse bronchitis, both sides, with redness of corn. Corn soft and pulpy.</p>
<p>No. 1154 No specimen</p>	<p>7 yrs.</p>	<p>Grain of corn (maize)</p>	<p>Right main bronchus, 3 days</p>	<p>None</p>	<p>6 mm.</p>	<p>Swollen kernel, impacted</p>	<p>Side-curved</p>	<p>Flatwise beyond edge</p>	<p>Extraction Cure</p>	<p>6 min. 35 sec.</p>	<p>Oral</p>	
<p>Fbdy. 1014</p>  <p>1014</p>	<p>3 yrs.</p>	<p>Peanut kernel</p>	<p>Right stem bronchus below orifice of right upper lobe bronchus, 1 day</p>	<p>None</p>	<p>5 mm., aspirating</p>	<p>Swollen mucosa, obliterated forceps spaces, frangible foreign body</p>		<p>Over equator</p>	<p>Extraction Cure</p>	<p>3 min. 32 sec.</p>	<p>Oral</p>	<p>Subglottic tissue swollen, dimpled only a very small clink, apparently just sufficient in area to keep patient from getting cyanotic. Both bronchi swollen nearly shut. Foreign body removed. To be removed by bronchoscopy. Second bronchoscopy: Subglottic tissue swollen. Larynx infiltrated. Left main bronchus swollen, diminishing lumen to about a half normal diameter. No foreign body visible. All visible bronchial orifices swollen nearly shut. Diffuse bronchitis. Mucosa thick, red and velvety. Third bronchoscopy: Left bronchus swollen, from bilur-cation downward, lumen closing during expiration completely. Left inferior lobe bronchus swollen entirely shut at orifice of upper lobe bronchus. Impaction of lower lobe bronchus failed to reveal any remaining fragment of nut kernel.</p>

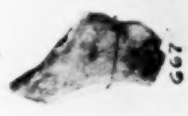

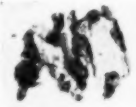
## BONES

Case Number and Illustration	Age	Foreign Body	Location	Anesthetin	Tube	Problem	Ex- cep- ta	Point of Seizure	Result	Time	Route	Comment
Fbdy. 637 	60 yrs.	Pork bone	Esophagus below cricopharyngeus, 3 days	None	9 mm.	None	Rotation	Rounded corner	Extraction Cure	1 min. 21 sec.	Oral	
Fbdy. 638 	43 yrs.	Lamb bone	Esophagus level of middle of thyroid cartilage, 2 days	None	9 mm.	Sharp pointed conoidal piece of bone with partial im- permeable and em- bedded	Rotation	One end	Extraction Cure	1 min. 23 sec.	Oral	
Fbdy. 639 	50 yrs.	Bone	Esophagus level of hyoid bone, 3½ hours	None	9 mm.	Bone wedge- l with both ends in cicatrical pockets	Rotation	One end	Extraction Cure	13 sec.	Oral	
Fbdy. 646 	24 yrs.	Fish bone	Overhanging larynx, 2 days	None	Laryn- goscope	None	Alligator	As pre- sented	Extraction Cure	10 sec.	Oral	



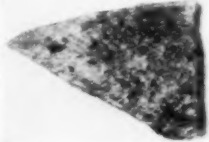


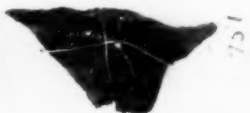

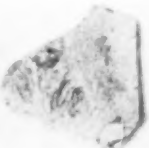
CHARTED EXPERIENCE OF FOREIGN BODY CASES.

1099

<p>Fbdy. 667</p>  <p>667</p>	<p>38 yrs.</p>	<p>Chicken bones</p>	<p>Esophagus below cricopharyngeal fold, 6 days</p>	<p>None</p>	<p>9 mm.</p>	<p>Finding bone in bed of ulcerated granulated tissue</p>	<p>Rotation</p>	<p>One end</p>	<p>Extraction Cure</p>	<p>7 min. 43 sec.</p>	<p>Oral</p>	<p>Granulating ulcer below cricopharyngeal fold.</p>
<p>Fbdy. 674</p>  <p>674</p>	<p>53 yrs.</p>	<p>Bone</p>	<p>Esophagus below cricopharyngeus, 2 days</p>	<p>None</p>	<p>9 mm.</p>	<p>Points buried in each lateral wall</p>	<p>Rotation</p>	<p>One end</p>	<p>Extraction Cure</p>	<p>3 min. 42 sec.</p>	<p>Oral</p>	<p>Localized esophagitis.</p>
<p>Fbdy. 685</p>  <p>685</p>	<p>34 yrs.</p>	<p>Bone</p>	<p>Esophagus below cricopharyngeus, 1 day</p>	<p>None</p>	<p>9 mm.</p>	<p>Rotation</p>	<p>Rotation</p>	<p>One end</p>	<p>Extraction Cure</p>	<p>2 min. 11 sec.</p>	<p>Oral</p>	

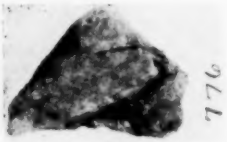


## BONES—Continued

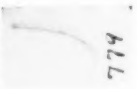
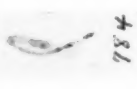

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 706  706	8 yrs.	Chicken bone	Larynx	None	7 mm.	None	Rotation	One angle	Extraction Cure	1 min. 30 sec.	Oral	Intense laryngitis. Slight erosion.
Fbdy. 714  714	44 yrs.	Fish bone	Esophagus, 2 days	None	9 mm.	Thin rib bone buried up to one point in mucosa	Side-curve.	Point	Extraction Cure	1 min. 20 sec.	Oral	
Fbdy. 729  729	4 yrs.	Beef bone	Esophagus, 14 days	None	Esophag- asacal specu- lum	Bone very tightly impacted below cri- copharyn- geus	Alligator	One angle	Extraction Cure	2 min. 53 sec.	Oral	Localized esophagitis. Mass of granulation tissue in right pyriform sinus.

Fbdy. 751		44 yrs.	Beef bone	Esophagus, 17 days	None	9 mm.	Extremely tight impaction of a very large foreign body below cricopharyngeus; long angles buried in mucosa and covered over with shreds of mucus membrane. Malpresentation.	Rotation	Lesser diameter after version	Extraction 14 min. 36 sec.	Oral	Esophageal walls ulcerated.
Fbdy. 752		30 yrs.	Pork bone	Esophagus, 3 days	None	9 mm.	Malpresentation	Rotation		Extraction 37 min. 22 sec.	Oral	Two small black slender objects attached to a tough grey membranous-looking substance were found below the cricopharyngeus. The slender black substances looked like a suture in the esophageal wall. After removing the foreign tissue and the little slender objects, a number of small splinter-like black fragments were seen in the copious, tough, glairy secretions. The site from which the foreign body was removed was eroded and very inflammatory.
Fbdy. 760		61 yrs.	Bone	Esophagus below cricopharyngeus, 5 days	None		None	Rotation		Extraction 39 sec.	Oral	



93 760



## BONES—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes-thetic	Tube	Prob-lem	For-cep	Point of Suture	Result	Time	Route	Comment
Ebdy. 776 	46 yrs	Bone	Esophagus, 11 days	None	9 mm.	Transfixed malpres-entation	Rotation		Extraction Cure	3 min. 8 sec.	Oral	Bone transfixed crosswise just below point where in-spiratory opening of the lumen of the thoracic esophagus took place (this normally makes place). Patient had a goitre which caused some compression stenosis of the cervical esophagus.
Ebdy. 777 	46 yrs.	Fish bone	Left tonsil about 3 hours						Extraction Cure			
Ebdy. 778 		Fish bone	Laryngopharynx						Extraction Cure			

<p>Fbdy. 779</p>  <p>779</p>	<p>15 yrs. Fish bone</p>	<p>Left tonsil, 1 day</p>				<p>Extraction Cure</p>	
<p>Fbdy. 784</p>  <p>784</p>	<p>32 yrs. Fish bone</p>	<p>Laryngopharynx 1 day</p>			<p>Extraction Cure</p>		
<p>Fbdy. 800</p>  <p>800</p>	<p>51 yrs. Fish bone</p>	<p>Lingual tonsil, 1 day</p>			<p>Extraction Cure</p>		

## BONES—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 814 	35 yrs.	Fish bone	Left tonsil						Extraction Cure			About 40 cases of foreign body in the tonsil have been omitted. These were chiefly costal bones of fish and birds, spicules of bones, tooth-brush bristles, and other slender sharp objects.
Fbdy. 817 	10 yrs	Chicken bone	Esophagus just above level crossing left bronchus, 4 days	None	9 mm.	Pathology	Rotation		Extraction Cure	30 sec.	Oral	Rather severe degree of acute esophagitis, not calized particularly to the neighborhood of the foreign body. Fbdy. lodged considerably below usual location of fbdes. in esophagus.

Fbdy. 820		820	37 yrs.	Fish bone	Esophagus, 1 day	None	9 mm.	None	Side-curved	One point	Extraction Cure	2 min. 54 sec.	Oral	Bone had penetrated esophageal wall to a depth of about half the length of bone.
No. 853 No specimen	37 yrs.	Fish bone	Tonsil, 1 day	None							Extraction Cure			
Fbdy. 857		857	72 yrs.	Bone	Esophagus, 3 days	None	9 mm.	Trans-fixed	Rotation		Extraction Cure	3 min. 40 sec.	Oral	Bone transfixed crosswise with left end perforating deeply into left latero-posterior wall.


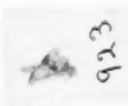
## BONE S.—Continued



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 861  861	44 yrs.	Pork bone	Right main bronchus at orifice of upper lobe bronchus, 4 days	Local	9 mm.	Malpresentation	Rotation		Extraction Cure	35 sec.	Oral	Greater transverse diameter of bone was in the anteroposterior plane.
Fbdy. 863  863	70 yrs.	Fish bone	Hypopharynx	None					Extraction Cure			
Fbdy. 867  867	61 yrs.	Bone	Right middle lobe bronchus obstructing lower lobe bronchus, about 1 year	Local	7 mm.	Pathology from 1 yr sojourn	Sidescurved	Flatwise near end	Extraction Cure	7 min. 40 sec.	Oral	Right main bronchus obstructed almost completely by granulation tissue. After partially removing these with forceps, the bone was stained red by oozing from granulations. Bone soft and friable at edge, turned with its greatest plane sagittally and withdrawn.






Fbdy. 879		56 yrs.	Bone (Duck)	Esophagus below cricoid cartilage, 4 days	None	9 mm.	Sharp pointed foreign body	Rotation	Near one end	Extraction Cure	43 sec.	Oral	Convexity of bone to left, points to right.
Fbdy. 883		32 yrs.	Fish bone	Laryngopharynx	None					Extraction Cure			
Fbdy. 895		13 yrs.	Fish bone	Left arytenoid eminence	None					Extraction Cure		Oral	




## BONE S.—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For-cups	Point of Suture	Result	Time	Route	Comment
Fbdy. 902 	37 yrs.	Beef bone	Esophagus below cricopharyngeus, 14 days	Local to phar- ynx	9 mm.	Bone firmly wedged	Rotation	Flatwise	Extraction Cure	1 min. 20 sec.	Oral	Bone quadrangular and quite thick. Localized esophagitis.
No. 910 No specimen	41 yrs.	Fish bone	Right tonsil	None					Extraction Cure		Oral	
Fbdy. 923 	31 yrs.	Fish bone	Right tonsil	None					Extraction Cure		Oral	
No. 938 No specimen	36 yrs.	Fish bone	Glossoepiglottic sulcus	None					Extraction Cure		Oral	




<p>Fbdy. 970</p> 	<p>3 yrs.</p>	<p>Chicken bone</p>	<p>Subglottic trachea, 14 days</p>	<p>None</p>	<p>Swollen esoph- geal folds</p>	<p>Slits- curved</p>	<p>Extraction 5 min. 50 sec</p>	<p>Oral</p>	<p>Fbdy. found at 27 cm. from upper alveolus in pharynx. It was stomped of the esoph- agus; lumen of narrowing will not admit 10 mm. esophagoscope (normal adult size). Slight dilata- tion of the narrowing line. The dilatation gesta a probability of the narrowing having been present for a long time; the narrowing is at the arch of the aorta, the positions of which are quite constant and are more so than would be expected in a man of this age and type. The piece of oyster shell of this size probably would not lodge in a perfectly normal esophagus.</p>
<p>Fbdy. 982</p> 	<p>70 yrs</p>	<p>Portion oyster shell</p>	<p>Esophagus, 4 days</p>	<p>None</p>	<p>10 min.</p>	<p>Slits- curved</p>	<p>Extraction 5 min. 50 sec</p>	<p>Oral</p>	<p>Intense laryngitis with swelling of ventricular bands and cords. Gran- ulation tissue in the larynx and posteriorly close to the bone on deep inspira- tion. Bone found in the sagittal plane just far enough below the cords to permit of the cords being raised and the bone was visible only on in- spiration. It did not pre- vent approximation and contact of cords with each other. Fbdy. seized on inspiration. The in- crease in tension due to excursion of the mucosa by the roughened edges of the bone was rapidly in- creasing the obstruction.</p>



## BONES—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Case 983 Fbody. 983 	46 yrs.	Beef bone	Esophagus below cricopharyngeus, 2 days	None	9 mm.	Transfixed sharp, through body.	Rotation	Left end	Extraction Cure	9 min. 25 sec.	Oral	Bone transfixed crosswise.
Case 987 Fbody. 987 	49 yrs.	Chicken bone	Esophagus just below cricopharyngeal fold, 1 day	None		Sharp edges	Rotation	Right end	Extraction Cure	1 min. 47 sec.	Oral	Bone found with the edge presenting, the greater plane of the bone being in the coronal plane of the body.
Case 1132 Fbody. 1132 	26 yrs.	Fish bone	Esophagus at level of first thoracic vertebra, 1 day	None	9 mm.	Point buried	Side-curved	Sharp end after freeing	Extraction Cure	4 min. 10 sec.	Oral	Slight laceration of esophageal wall at point of entrance of bone embedded slightly in right lateral wall.

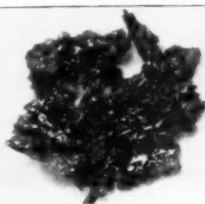
<p>Fbdy. 1155</p> 	<p>55 yrs.</p>	<p>Chicken bone</p>	<p>Esophagus below cricopharyngeus, 7 days</p>	<p>None</p>	<p>Forbes' speculum</p>	<p>Bone, run-fixed crosswise</p>	<p>Laryngeal aspirating</p>	<p>Extraction 2 min. 35 sec. Cure</p>	<p>Oral</p>	
<p>Fbdy. 1013</p> 	<p>55 yrs.</p>	<p>Veal bone</p>	<p>Esophagus just below cricopharyngeus, 2 days</p>	<p>None</p>	<p>Esophageal speculum</p>	<p>Transfixed; points buried</p>	<p>Plain</p>	<p>Slender end</p>	<p>Extraction 1 min. 57 sec. Cure</p>	<p>Oral</p> <p>Bone, slender, about 3 cm. long, transfixed lengthwise.</p>
<p>Fbdy. 1023</p> 	<p>29 yrs.</p>	<p>Chicken bone</p>	<p>Esophagus below crossing of left bronchus, 2 days</p>	<p>None</p>	<p>9 mm.</p>	<p>Prevention; avoidance of trauma</p>	<p>Plain</p>	<p>Flatwise covering sharp point</p>	<p>Extraction 1 min. 20 sec. Cure</p>	<p>Oral</p> <p>Greater plane of bone in coronal plane.</p>

## BONES—Continued


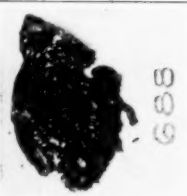
Case Number and Illustration	Age	Foreign Body	Location	Anes-thetic	Tube	Problem	For-ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1045 	27 yrs.	Fish bone	Esophagus	None	Esoph- ageal spec- ulum	Presenta- tion	Slide- curved	As pre- sented	Extraction Cure		Oral	
Fbdy. 1087 	44 yrs.	Pork bone	Esophagus below cricopharyngeus, 2 days	None	Esoph- ageal spec- ulum	Buried point	rota- tion	Near buried point	Extraction Cure	15 min. 9 sec.	Oral	About one cm. of pointed end buried in posterior wall.
Fbdy. 1101 	44 yrs.	Beef bone	Esophagus below cricopharyngeal fold, 1 week	None			Slide- curved	By one pointed end	Extraction Cure	4 min. 30 sec.	Oral	Long axis of bone cross- wise, point perforating right wall.

Fbdy. 1123  1125	35 yrs.	Chicken bone	Esophagus at level of cricoid of left bronchus, 1 day	None	9 mm.	Crosswise fixation; buried; avoidance of trauma	Rotation	Free and	Extraction 2 min. 7 sec.	Oral	Bone lodged long axis crosswise; end to right perforating into esophagus; end to left in contact but not perforating esophageal wall.
Fbdy. 1128  1128	27 yrs.	Bone	Left pleural cavity	None	Pleuroscope (7 mm. retrograde gastroscopy)				Extraction	Pleural (retrograde gastroscopy)	Small spicule of bone removed from pleural cavity during pleuroscopy for examination. Patient had a portion of projectile in left lung. See body. No. 1131.


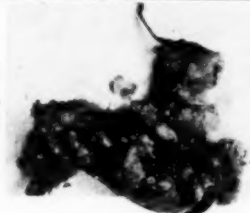

MEAT.

Fbdy. 661  661	71 yrs.	Meat	Esophagus at cricopharyngeal constriction	None	Esophagoscopy (ultrum)	None	Pancreas-treated		Extraction Cure	Oral	
---	---------	------	---	------	------------------------	------	------------------	--	-----------------	------	--

## MEAT—Continued

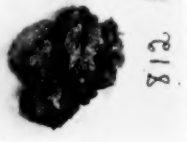

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 672 	66 yrs.	Meat	Esophagus below cricopharyngeal fold, 6 hours	None	Long esoph- ageal spec- ulum	Friable	Side- curved		Extraction Cure	5 min.	Oral	
Fbdy. 688 	40 yrs.	Meat	Esophagus both above and below cricopharyngeus, 12 hours	None	Esoph- ageal spec- ulum	Friable	Alligator		Extraction Cure		Oral	Meat found embedded in an old ulcerated infiltra- tion. Bleeding from gran- ulation tissue.


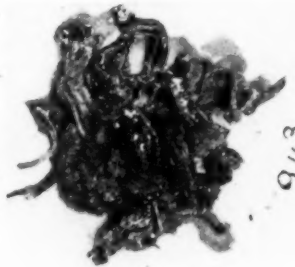
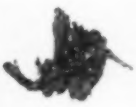


Fbdy. 765	 765	64 yrs.	Meat	In malignant stenosis of esophagus	None	9 mm.	Fragile	Side-curved			Oral	Removed during esophagoscopy for diagnosis.
Fbdy. 808	 808	12 yrs.	Bolus Meat	In old stricture of esophagus, 1 day	None	7 mm.	Meat soft and friable	Side-curved	Extraction Cure	Extraction 1 min. 2 sec.	Oral	Upper portion of mass of meat very soft and friable. This was removed first and then a good grasp was obtained on a tougher portion which came away in one portion measuring about 2 cm. in diameter by 7 cm. in length.
Fbdy. 811	 811	68 yrs.	Meat	In malignant stenosis of esophagus	None	9 mm.	None	Side-curved	Extraction Relief		Oral	Removed during esophagoscopy for diagnosis.


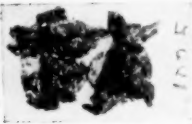
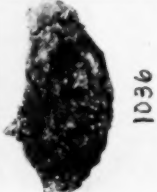
107

## MEAT—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For-cops	Point of Seizure	Result	Time	Route	Comment
Fbdy. 812 	27 yrs.	Meat	Esophagus	None	10 mm.	None	Side-curved		Extraction Relief		Oral	Case of preventriculolysis.
No. 821 No specimen	10 yrs.	Meat	In old stricture of esophagus. 3 days	None	7 mm.	Foreign body in tight stricture			Foreign body pushed in each esophagoscopically	5 min. 31 sec.		History of swallowing lye at 2 years of age. Inflammation of esophagus. Old stricture. Esophagus congested. Vessels crossing scar tissue; small erosions over large surface. Lumen of stricture readily admits No. 12 esophagoscope. Brought but tightens on No. 14 and No. 16 is gripped very tightly.
Fbdy. 826 	52 yrs.	Meat	Esophageal diverticulum	None	9 mm.	None	Side-curved		Extraction		Oral	Case of diverticulum. Removed during esophagoscopy for diagnosis.



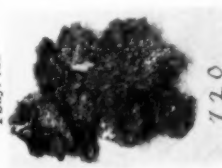
Fbdy. 845  845	54 yrs. Meat	In malignant stenosis of esophagus	None	9 mm.	None	Side-curved	Extraction			Removed during esophagoscopy for diagnosis but removal afforded relief.
Fbdy. 943  943	25 yrs. Meat	In old stricture of esophagus	None		Foreign body in old stricture	Side-curved	Extraction	Oral		Meat impacted in stricture of esophagus, at crossing of left bronchus.
Fbdy. 1001  1001	Meat	Esophagus	None	7 mm.	None	Side-curved	Extraction Relief		14 min. 17 sec.	Mass of stringy fibres of ham found impacted tightly in the grasp of the esophageal wall at the hiatus. Part of fibres were removed and the other part passed down into the stomach and esophagus ahead of the esophagoscope.

## MEAT—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1012 	46 yrs.	Meat	Esophagus, at suprasternal notch, 2 days	None	Esophageal speculum	Pulpy, friable foreign body without cohesion	Meat	Presenting part; numerous scizures	Extraction Cure	12 min. 37 sec.	Oral	Marked narrowing of esophageal lumen at level of suprasternal notch.
Fbdy. 1025 	41 yrs.	Meat	Esophagus, below cricopharyngeus, 1 day	None	Esophageal speculum	Pulpy, friable foreign body without cohesion	Plain	Presenting part	Extraction Cure	3 min. 3 sec.	Oral	After removal of meat, esophageus passed at very tight fit through the cervical esophagus into stomach. No scars visible.
Fbdy. 1036 	54 yrs.	Meat	Esophagus in cancerous stenosis	None	9 mm.	None	Side-curved	Presenting part	Extraction Relief		Oral	

No. 1032 No specimen	Ment	Esophagus (stricture)	None	7 mm.	Side- curved	Presenting part	Extraction Relief	5 min. 5 sec.	Oral	Mass of soft pulp found "corking" the lumen of the stricture. Severe degree of esophagitis observed throughout all region of stricture.
-------------------------	------	--------------------------	------	-------	-----------------	--------------------	----------------------	------------------	------	--


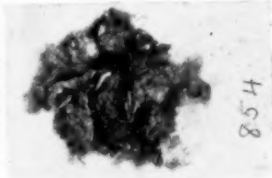

FOOD

Fbdy. 698 	4 yrs.	Chewing gum	Strictured esophagus (old lesion)	None	7 mm.	None	Plain		Extraction Cure		Oral	
Fbdy. 700 	4 yrs.	Particles food	Strictured esophagus (old lesion)	None	7 mm.	None	Plain		Extraction Cure	43 sec.	Oral	Intense esophagitis local- ized in neighborhood of stricture.
Fbdy. 720 	4 yrs.	Dry custard	Strictured esophagus (old lesion)	None		None	Side- curved		Extraction Cure		Oral	

## FOOD—Continued

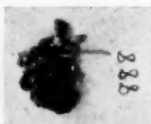



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- eign	Point of Seizure	Result	Time	Route	Comment
Fbdy. 795	59 yrs.	Bolus of food; meat, bones, lettuce, seeds, lettuce, onion	Esophagus 4 cm. below crossing of trachea; lower lobe bronchus	None	10 mm. gastro-scope	Exceedingly irritable condition of meat	Side- viewed and meat		Extraction Cure	1 hr. 25 min	Oral	Fbdy. continued to come out until finally a tumorous portion was uncovered and seized. No lesion of esophagus visible to account for the lodgment except that the esophagus is dilated to several times its normal diameter at the localization of the foreign body. Imperfect mastication, many teeth absent.






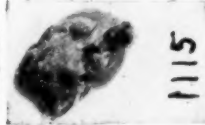
Fbdy. 804 	65 yrs.	Three peas	In malignant stenosis of esophagus	None		Side-curved and meat		Extraction Cure of fbdy. ob- struction		Removed during esophago- scopy for diagnosis but removal afforded relief
Fbdy. 854 	47 yrs.	Portions fish	In old stricture of esophagus, 3 days	None	7 mm.	Side-curved and meat		Extraction Cure	20 min. 55 sec.	Fish found in electrical channel packed tightly in a column about 4 cm. deep. After removal of the fish, 7 mm. esophago- scope was inserted without difficulty. Just the electrical current above it was a large, white, pitted scar looking like a vaccination scar, extending about 2 cm. laterally and about 5 cm. upward and downward.
Fbdy. 858 	4 yrs.	Pieces orange skin	In strictured esophagus	None		Finding in folds and cicatrices		Extraction Relief	2 min. 48 sec.	Congenital stricture.

Note: In all cases of foreign body in a strictured esophageal human the patient was discharged cured of foreign body obstructions; but in many cases the word "cure" was omitted from the records because of the organic condition. Most of the strictures were from swallowing lyc; some of them were from previous lymphatic suppurations.

## FOOD—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- e- caps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 888 	6 mos.	Meat	In strictured esophagus	None		Finding fbdy. in folds and cicatrices			Extraction	3 min. 35 sec.	Oral	
Fbdy. 897 	22 mos.	Food	In strictured esophagus	None		Finding fbdy. in folds and cicatrices			Extraction	2 min. 5 sec.	Oral	
Fbdy. 903 	2 yrs.	Paper or fabric	In strictured esophagus	None		Finding fbdy. in strictured lumen			Extraction	1 min. 50 sec.	Oral	
Fbdy. 908 	2½ yrs.	Piece orange skin	In strictured esophagus (Con- genital stenosis)	None		Finding fbdy. in lumen of narrow stricture			Extraction	1 min. 50 sec.	Oral	Fbdy. found fitted closely in funnel of stricture. No esophagitis. Orange peel was acting inter- mittently as a check valve.




Fbdy. 1038 	62 yrs.	Food	Esophagus	None	9 mm.	None	Fenestrated		Extraction Relief		Oral	Removed during esophagoscopy for diagnosis (cancer). Relief afforded.
Fbdy. 1049 		Food	Esophagus	None	7 mm.	None	Slide-curved		Extraction Relief	3 min. 20 sec.	Oral	Grain of corn and mass of meat found compacted in esophagus just above hiatus. High degree of esophagitis.
Fbdy. 1077 		Food	Esophagus (strictured)	None	7 mm.	None	Slide-curved		Extraction Relief	1 min. 25 sec.	Oral	
Fbdy. 1115 	5 yrs.	Orange pulp	Esophagus (strictured)	None	7 mm.	Foreign body located between two strictures	Slide-curved		Extraction Relief		Oral	

## FOOD—Continued



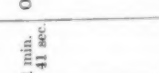
Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Suture	Result	Time	Route	Comment
Fbdy. 1129  1129	22 yrs.	Food	Esophagus middle third, 3 days	None	9 mm.	Pulpy mass lacking cohesion	Side-curved		Extraction	3 min. 10 sec.	Oral	Patient has marked stenosis at point of lodgment. Second lodgment of food.
Fbdy. 1146  1146	5 yrs.	Orange pulp	Esophagus	None	7 mm.	Lodged between two strictures	Side-curved		Extraction	3 min. 4 sec.	Oral	Removed during esophagoscopy bougienage. Case of lye stricture.

## BUTTONS



Fbdy. 693  693	2 yrs.	Button	Esophagus at crossing of left bronchus, 2 days	None	7 mm.	Hard slippery surface	Rotation	Minor axis	Extraction Cure	37 sec.	Oral	
---	--------	--------	--	------	-------	-----------------------	----------	------------	--------------------	---------	------	--




CHARTED EXPERIENCE OF FOREIGN BODY CASES.

1125

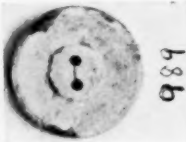
Fbdy. 705		13 mos.	Button	Esophagus below cricopharyngeus border of sternum, 2 days	None	Laryngoscopic	Hard slippery surface	Side-curved	Flatwise	Extraction 28 sec. Cure	Oral	Masses of whitish looking food surrounding button which was embedded at the upper thoracic aperture.
Fbdy. 755		14 mos.	Portion button	Right bronchus, 53 days	None	5 mm.	Transfixed and "hooked" at orifice of right upper lobe bronchus	Side-curved	End after version	Extraction 14 min. 18 sec. Cure	Oral	Subglottic tissue much swollen. White object seen in right bronchus, transfixed at the orifice of the upper lobe bronchus. Traction with forceps demonstrated that the far end must be hooked around the corner. Manipulation with forceps finally succeeded in turning foreign body until the hook-shaped end was disengaged and the long axis of the bronchus corresponded to the long axis of the foreign body when the foreign body came away easily.
Fbdy. 759		3 yrs.	Button	Esophagus below cricopharyngeus, 20 days	None	Esophagoscopy	Avoidance of false passages	Alligator	Flatwise	Extraction 1 min. 41 sec. Cure	Oral	Blood coming from many lacerations in the esophagus. Severe esophagitis.

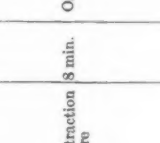
## BUTTONS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 852 	14 mos.	Button	Esophagus below cricopharynx, 14 days	None	7 mm.	Button covered over by folds of edematous esophageal mucosa which required considerable manipulation to uncover button for placement of forceps	Plain	Flatwise	Extraction Cure	3 min. 30 sec.	Oral	Traumatic esophagitis present from which there was free bleeding.
Fbdy. 865 	3 yrs.	Campalgn button	Esophagus below cricopharynx, 2 days	None	6 mm. Esophageal speculum	Rigid pin, point upward, piercing posterior esophageal wall	Rotation	Left edge to produce version	Extraction Cure	2 min. 4 sec.	Oral	Disc of pin grasped as far to left side as possible and rotated half around to prevent trauma by point of pin. Esophageal speculum used to make side pressure so as to turn button.

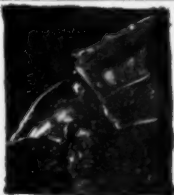
<p>Fbdy. 893</p>  <p>893</p>	<p>6 yrs.</p>	<p>Button</p>	<p>Esophagus below cricopharyngeus, 2 days</p>	<p>None</p>	<p>Esophag- o- spec- ulum (Forbes)</p>	<p>Button very tightly wedged; covered with cri- copharyn- geal fold</p>	<p>Down- jaw</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>1 min. 33 sec.</p>	<p>Oral</p>											
<p>Fbdy. 894</p>  <p>894</p>	<p>18 mos.</p>	<p>Button</p>	<p>Esophagus below cricopharyngeus, 8 days</p>	<p>None</p>	<p>Laryn- go- scope</p>	<p>Button very tightly wedged</p>	<p>Down- jaw</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>1 min. 1 sec.</p>	<p>Oral</p>											<p>Button found with small erosion at base of left antero; large end deeply embedded in folds.</p>
<p>Fbdy. 971</p>  <p>971</p>	<p>16 mos.</p>	<p>Patent button ("Batch- elors")</p>	<p>Esophagus about 2 in. below crico- pharynx, about 5 months</p>	<p>None</p>	<p>Laryn- go- scope</p>	<p>Presenta- tion</p>	<p>Alligator</p>		<p>Extraction Cure</p>	<p>1 min. 35 sec.</p>	<p>Oral</p>											

## BUTTONS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 989  989	3 yrs.	Button	Thoracic esophagus at crossing of left bronchus, 9 days	None	7 mm.	Preser- ta- tion	Plain	Flatwise	Extraction Cure	1 min. 15 sec.	Oral	
Fbdy. 995  995	2 yrs.	Button	Esophagus below cricopharyngeus, 23 days	None	7 mm.	Pathology from 23 days' sojourn	Plain	Flatwise	Extraction Cure	3 min. 30 sec.	Oral	Patches of granulation which bled very freely. Button buried in inflammatory folds of esophageal wall.
Fbdy. 996  996	3 yrs.	Button	Esophagus, 4 months	None	7 mm.	Pathology from 4 months' sojourn	Plain	Flatwise	Extraction Cure	4 min. 55 sec.	Oral	Button hidden by folds of esophageal wall and buried in a swollen mass of edematous tissue.

Fbdy. 1091		14 mos.	Button	Esophagus below cricopharyngeus, 19 days	None	7 mm.	Prevention of dyspnea	Plain	Flatwise	Extraction 8 min. Cure	Oral	Bleeding granulations in the esophagus and at the cricopharyngeal fold. Button embedded in posterior esophageal wall.
------------	---	---------	--------	--	------	-------	-----------------------	-------	----------	------------------------	------	---




GLASS

No. 690 No specimen	3 yrs.	Glass lens from flash- light	Esophagus below cricopharyngeus, 3 days	None	7 mm.	Smoothness and shape of body.; lack of re- sistance below; closeness of fit of walls	Plain	Fbdy. al- lowed to recede in- to stom- ach; re- covered in stools	34 min. 39 sec. 24 min. 18 sec.	Right and left walls of esophagus showed gran- ular ulceration at site of lodgment of fbdy.
Fbdy. S18 	18 mos.	Portion glass breast pump	Esophagus below cricopharyngeus, 14 days	None	7 mm.	Fragile, smooth, hard body, embedded at sharp, cutting edges	Special jaws made to fit body.	Extraction 6 min. Cure	Oral	Glass found tightly im- pacted between cricoid and posterior wall sur- rounded by granulations which oozed blood, almost black, very freely. Sloughing eso- phagitis. Child very toxic from sloughing esophagitis.

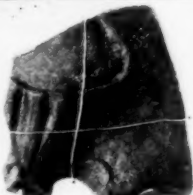

818

121



## DENTAL AND SURGICAL OBJECTS

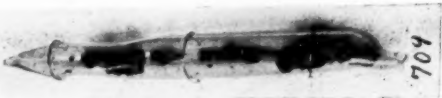
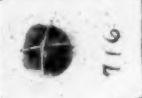
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
No. 643 No specimen	21 yrs.	Dental Instrument	At upper lobe bronchus, 15 days	Local	9 mm.	Upper lobe bronchus "around the corner"	Upper lobe forceps		Extraction Cure	21 min.	Oral	
Fbdy. 645 	6 yrs.	Electric bulb from mouth gag	Left bronchus, 6 days	None	5 mm.	Tight impaction	Side-curved	By threaded base	Extraction Cure	1 min. 9 sec.	Oral	Localized bronchitis.
Fbdy. 653 	43 yrs.	Band of gold crown	Left lower lobe bronchus at orifice of posterior branch, 13 days	Local	9 mm.	None	Side-curved	Both edges of presenting portion	Extraction Cure	1 min. 49 sec.	Oral	Intense laryngitis. Exudate. Granulation tissue.
Fbdy. 666 	45 yrs.	Dental Instrument	Right inferior lobe bronchus, 10 days	Local	9 mm.	Fbdy. in small branch "around the corner"	Side-curved	Finding in small bronchus	Extraction Cure	10 min. 1 sec.	Oral	Very slight localized bronchitis.




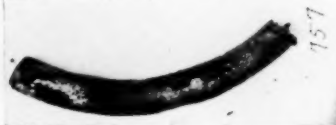
 <p>Fbdy. 669</p> <p>669</p>	48 yrs.	Portion of tooth plate	Esophagus at level lower margin thyroid cartilage, 1 week	None	9 mm.	Tight impaction; inflamed; abscess; sensation	Side-curved	Near one end after version	Extraction Cure	14 min. 39 sec.	Oral	Sloughing esophagitis. Fistula leading backward.
 <p>Fbdy. 675</p> <p>675</p>	44 yrs.	Gold three-tooth bridge	Right main bronchus at orifice of upper lobe bronchus, 5 days	Local	9 mm.	Necessity for opening at least diameter	Side-curved	Presenting gold band	Extraction Cure	31 sec.	Oral	Localized bronchitis. Pus. Size of fishy 3 cm. x 1.5 cm. Circumference 4 cm.



## DENTAL AND SURGICAL OBJECTS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 679 	7 yrs.	Intubation tube	Left main bronchus, 18 mos.	None	5 mm.	Hollow object fitting tightly	Mosquito	One blade of forceps inside, other blade outside	Extraction Cure	3 min. 59 sec.	Oral	Mucosa of larynx intensely inflamed, tracheal and bronchial mucosa also. Left bronchial orifice larger than right. Large quantities of foul, pinkish pus removed by sponge. Patient died at bottom of dilatation of left bronchus.
Fbdy. 683 	18 yrs.	Dental instrument	Posterior branch left inferior lobe bronchus, 1 mo. 9 days	None	7 mm.	Small object in all branch bronchus	Silico-curved	Point	Extraction Cure	2 min. 2 sec.	Oral	Trachea red and inflamed. Mucosa of trachea and pharyngeal mucosa; left bronchus high degree of inflammation with adherent globular masses of thick tough secretion.

<p>Fbdy. 709</p> 	<p>51 yrs.</p>	<p>Portion of tooth</p>	<p>Right lower lobe bronchus, 4 1/2 years</p>	<p>Local</p>	<p>7 mm.</p>	<p>In abscess above fibrous stricture</p>	<p>Side-curved</p>	<p>Extraction</p>	<p>1 hr. 31 in. 1 hr. 17 min. 25 min. 26 min</p>	<p>Oral</p>	<p>Four bronchoscopies done 4 in. in. M. removed disease, secondary to pulmonary abscess caused by presence of foreign body.</p>
<p>Fbdy. 716</p> 	<p>49 yrs.</p>	<p>Amalgam tooth filling</p>	<p>Lung tissue of right lower lobe, 7 months</p>	<p>Local</p>	<p>5 mm.</p>	<p>Extreme depth of foreign body in lung tissue</p>	<p>Plain</p>	<p>Extraction Cure</p>	<p>41 min. 38 sec. 23 min. 38 sec.</p>	<p>Oral</p>	<p>Lower lobe bronchus in tensely inflated. Pus coming up from small median branch.</p>




## DENTAL AND SURGICAL OBJECTS—Continued

Case Number and Illustration	Age	Foreign in Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 753 	14 yrs.	Tooth	Right main bronchus, 23 days	None	7 mm.	Abscess			Extraction Cure	19 min. 49 sec.	Oral	Right main bronchus swollen almost shut; outline elliptical; axis from 10 to 4 o'clock; walls collapsed to contact during expiration, opening again during inspiration. Middle lobe bronchus, orifice completely closed by swelling and compression of the tooth; tooth found, crown upward, in stem bronchus, the top of the crown being flush with the gap between the middle lobe bronchus and orifice and inferior lobe bronchial orifice. As soon as tooth was dislodged by sponging away the blood-stained secretion, large quantities of purulent pus were liberated, the patient coughed, and the same foul odor noted on the breath at the start of bronchoscopy.
Fbdy. 757 	8 yrs.	Portion tracheotomy tube	Trachea, 2 days	None	6 mm.	Hollow object	Silenced by edge	One jaw inside, other outside, of end	Extraction Cure	1 min. 17 sec.	Oral	Patient wearing tracheotomy tube for papilloma 2 years, with no change in size. At bronchoscopy larynx found perfectly patulous. No sign of papillomata anywhere. Mucosa of larynx in subglottic region normal. Glottis normal. Upper end of cannula seen in trachea. No bubbling to indicate the passage of air through the cannula. Ragged edge of foreign body drawn into mouth to protect the tissues during withdrawal.



<p>Fbdy. 773</p> 	<p>45 yrs.</p>	<p>Portion tooth plate</p>	<p>Left main bronchus, 4 months</p>	<p>Local</p>	<p>7 mm.</p>	<p>None</p>	<p>Side-curved</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>23 sec.</p>	<p>Oral</p>	<p>Foreign body lying free in left bronchus above orifice of upper lobe bronchus. Slight degree of localized reaction only. Right bronchus and trachea looked normal.</p>
<p>Fbdy. 840</p> 	<p>31 yrs.</p>	<p>Root of tooth</p>	<p>Right stem bronchus, below largest posterior branch</p>	<p>Local</p>	<p>7 mm.</p>	<p>Acute inflammatory stenosis</p>	<p>Side-curved</p>	<p>Presenting portion</p>	<p>Extraction Cure</p>	<p>18 min. 7 sec.</p>	<p>Oral</p>	<p>Trachea and carina inflamed with patchy red color; right stem bronchus below upper lobe bronchus very red and the mucosa swollen; granulations filled stem bronchus below main stem bronchus. Right inferior lobe bronchus bronchiectatic. Considerable quantities of pus and blood kept constantly coming up from the inferior lobe bronchus. Bronchus characterized as purulent and pus characteristically asphyctic but not quite so foul as in most foreign body cases of prolonged sojourn.</p>

## DENTAL AND SURGICAL OBJECTS—(Continued)

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Suture	Result	Time	Route	Comment
Fbdy. 844 	22 yrs.	Tooth	Branch of the right inferior lobe bronchus, 51 days	Local	7 mm.	Abscess	Plain	Presenting end	Extraction Cure		Oral	<p><i>First attempt:</i> One branch of middle lobe bronchus and one branch of inferior lobe bronchus swollen shut. The small lumen was filled with pus in each case. Both swollen bronchial orifices were dilated with forceps used during expansion. The orifice of both bronchi contained granulations, and in the middle lobe bronchus there was a hard substance which could not be forced but it was not free to move. Time: 34 min. 27 sec.</p> <p><i>Second attempt:</i> Orifice of branch of middle lobe bronchus seemed to be granulated. In the inferior lobe bronchus the anterior branch which before was seen to be swollen shut, was more patulous though occupied by granulation tissue. This was removed and the orifice dilated with forceps. Teeth found with one end presenting; this was seized and tooth found to be freely movable and came away on very gentle traction. 6 min. 25 sec.</p>
Fbdy. 855 	63 yrs.	Gold crown	Right lower lobe bronchus, 12 days	Local	7 mm.	Rough sharp edge of open end upward	Plain	Conoidal end after version	Extraction Cure	27 min. 55 sec.	Oral	Unfavorable presentation converted to favorable one by version.

 <p>Fbdy. 882</p>	30 yrs.	Gold crown	Right main bronchus at orifice of middle lobe bronchus, 2 days	Local	9 mm.	Tight impaction. Version required	Side-curved	Outside of band after version	Extraction Cure	1 min. 1 sec.	Oral	Mucosa of entire tracheo-bronchial tree much reddened. No granulations present. Some mucus.
 <p>Fbdy. 887</p>	28 yrs.	Tooth plate	Esophagus, 7 days	None	9 mm.	Version required	Plain	One end after version	Extraction Cure	2 min. 11 sec.	Oral	No trauma of esophagus. No reaction around tooth plate.
 <p>Fbdy. 901</p>	7 yrs.	Spring catch from tonsil forceps	Right lower lobe bronchus, 15 days	None	3 mm.	Barb-like points preventing withdrawal	Side-curved	Presenting end	Extraction Cure	24 min. 16 sec.	Oral	Barbs protected by tube-mouth during withdrawal

## DENTAL AND SURGICAL OBJECTS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
No. 934 No specimen	27 yrs.	Tooth plate	Esophagus, 22 days	None	9 mm.	Unfavorable presentation. Two upward projecting points			Guided in- to stom- ach. Cure	27 min. 24 sec.		Subsequent x-ray studies show the position of foreign body in gastro-intestinal tract.
Fbdy. 937 	24 yrs.	Tooth plate	Esophagus, 3 days	None	9 mm.	Two upward projecting points	Side-curved	One point after removal of other point	Extraction Cure	3 min. 9 sec.	Oral	Point broken off with side-curved forceps to permit version.
No. 965 No specimen	51 yrs.	Partial tooth plate	Esophagus below cricopharyngeus, 19 days	None	Short esophago-scope	Firm impaction	Relation	By one "end" after version	Extraction Cure	4 min. 45 sec.	Oral	Unfavorable presentation required version.
Fbdy. 966 	29 yrs.	Tooth	Right stem bronchus, 8 months	Local		Firm impaction; only one space; other one created	Plain		Extraction Cure	2 min. 55 sec.	Oral	Large mass of granulation tissue in right stem bronchus just below the orifice of the middle lobe bronchus. The mass had wall stenosed apparently by perichondritis, the proximal swelling being quite firm.



Fdy. 978



50 yrs

Surgical  
drainPleural cavity,  
2 days

None



Retro-  
grade  
esoph-  
ago-  
scope  
used  
as a  
pleuro-  
scopeSilbo  
curved

End

Extraction 2 min.




Drain found about four inches from skin surface in a clean pocketed-off portion of the pleural cavity. After removal of body, pleuroscope again inserted but cavity not expected but not thor- oughly explored. The in- terior so far as seen was perfectly healthy and free from adhesions ex- cept those that formed the boundaries of the cavity. Granulations were nowhere visible in the explored region.




## DENTAL AND SURGICAL OBJECTS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1032 	34 yrs.	Tooth plate	Esophagus just below cricopharyngeus	None	9 mm.	Required version	Rotation	By left prong	Extraction Cure	2 min. 8 sec.	Oral	
1032 	24 yrs.	Tooth plate	In hypopharynx, lower part on posterior wall below cricopharyngeus into esophagus	None	Forbes	Two hooks embedded, one in pharyngeal wall, one in esophageal wall, opposite directions	Rotation		Extraction Cure	1 min. 5 sec.	Oral	Ruthless withdrawal would have inflicted wounds, possibly fatal trauma.
Fbdy. 1074												


 <p>Fbdy. 1107</p> <p>1107</p>	42 yrs.	Tooth plate	Esophagus below cricopharyngeus	None	Forbes	Required version	Rota- tion	Apex after version	Extraction Cure	2 min. 50 sec.	Oral	
 <p>Fbdy. 1130</p> <p>1130</p>	18 yrs.	Tooth-plate brace	Esophagus above suprasternal notch	None	Forbes	Required partial version	Mathieu	By left prong	Extraction Cure	3 min. 56 sec.	Oral	
 <p>Fbdy. 1094</p> <p>1094</p>	10 yrs.	Portion of wire dental brace	Lying across pharynx imme- diately behind epiglottis	None	None	Prevention of escape downward	Angular	By irregu- lar end	Extraction Cure		Oral	

## DENTAL AND SURGICAL OBJECTS—Continued



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1106  1106	17 yrs.	Dental brace	Left stem bronchus below orifice of upper lobe	Local	7 mm.	Sharply pointed; transfixed	Plain	Sharp point	Extraction Cure	7 min. 14 sec.	Oral	
Fbdy. 1121  1121	12 yrs.	Tooth	Right stem bronchus, 3 months	None	6 mm.	Pathology: absence of forceps spaces	Plain	Antero-posterior diameter	Extraction Cure	13 min. 53 sec.	Oral	
Fbdy. 1136  1136	20 yrs.	Tooth	Right stem bronchus, orifice of middle lobe, 66 days	Local	8 mm.	Pathology: absence of forceps spaces	Plain	Near one end	Extraction Cure	58 sec.	Oral	
Fbdy. 1153  1153	20 yrs.	Tooth filling	Left inferior lobe bronchus, 3 mos.	Local	7 mm. aspirating	Presentation in granulations and pus	Side-curved	Flatwise	Extraction Cure	2 min. 15 sec.	Oral	Granulations in inferior lobe bronchus at orifice of left upper lobe bronchus.

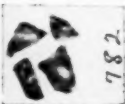

Fbdy. 841  841	6 yrs.	Cartridge blank	Left lower lobe bronchus, 12 days.	None	7 mm.	No furrows spaces; proximal end of bronchus stenosis	Specialy formed bronchus, thin plain forceps	Periphery after removal location above annular cicelma	Extraction 20 min. 35 sec. Cure 1 min. 33 sec.	Oral	Cartridge in left main bronchus with swelling of swelling proximally, hiding all of the end of primer except central portion which is seen to be indented.
Fbdy. 876  876	17 yrs.	Bullet (Penetrating gunshot)	In lung tissue of right lower lobe, 1 yr. 4 mos.	Local		Bullet in lung tissue, not in bronchus; anterior to bronchus, the bronchial wall intervening; penetrating into lung tissue, the lung tissue avoids the avoidance of blood vessels, and the tracing of bullet from cardiac cord in penetration of bullet		Heavy, plain	Extraction 36 min. 51 sec. Cure 35 min.		Bullet had entered by penetration through chest wall. First bronchoscopy for localization. Radiographs taken with instrument in position indicated to determine relation of bullet to stem bronchus. Fbdy. removed at second bronchoscopy. No hemorrhage. No elevation of temperature. Discharged cured 6 days later. Wound of entrance in back, between 7th and 8th ribs 8 cm. to right of spine.
Fbdy. 925  925	10 yrs.	Rifle bullet	Right stem bronchus, entering orifice of inferior lobe bronchus, 46 days	None	6 mm.	None	Slide-curved	By stem of bullet casting	Extraction 1 min. 1 sec. Cure	Oral	Great flood of pus from drowned lung "uncorked" by removal of bullet.

## AMMUNITION—Continued


Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1034  1034	9 yrs.	Cartridge shell	Left upper lobe bronchus, 15 days	None	7 mm.	Findings	Plain	By rim	Extraction Cure	14 min. 10 sec.	Oral	Pus coming from left bronchus. After remov- ing this bronchus, the scope was inserted deeply into left bronchus enabled remov- al of more pus but foreign body not seen. Broncho- scope then inserted into right bronchus which was seen to contain bronchus scope. Bronchus in left bronchus. Cartridge im- paled projecting from ori- fice of left upper lobe into lumen of stem bronchus.

## TOYS




Fbdy. 689  689	3 yrs.	Metal dog	Esophagus below cricopharyngeus, 8 days	None	7 mm.	Four bur- ied legs, tail and three legs deeply an- chored in esopha- geal wall.	Sides curved	Hind leg, tail being covered by tube- mouth during with- drawal	Extraction Cure	4 min. 30 sec.	Oral	Cricopharyngeal fold swell- ing. Just below it large folds of edematous poly- poid-looking mucosa. In- tense inflammatory re- action. Mucosa bleed- ing.
Fbdy. 721  721	4 mos.	Glass marble	Esophagus below cricopharyngeus, 4 hours	None	7 mm.	Smooth, hard globe	Safety- pin clasper used as ne- chan- ism for spoon		Extraction Cure	4 min. 51 sec.	Oral	

 <p>Fbdy. 782</p>	15 mos.	Portion glass from Christmas ball	Larynx, 4 1/2 months	None	Laryn- go- scope	Pathology from 4 months' sojourn. Fragile	Alligator		Extraction 39 sec. Cure	Oral	<p>Pieces of glass about 1 1/2 square cm. in area seen on edge precisely in the sagittal plane looking like a white thread. Fragment was broken into three pieces against the laryngoscope after emergence from mouth.</p>
 <p>Fbdy. 914</p>	4 yrs.	Toy opera glasses	Esophagus below cricopharyngeus, 1 day	None	Esoph- agesc- ulum	Presenta- tion	Alligator	Presenting part (one "ocular" end)	Extraction 45 sec. Cure	Oral	


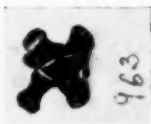
## TOYS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Suture	Result	Time	Route	Comment
Body: 915 	8 yrs.	Toy jack	Mediastinum out- side esophageal wall at crossing of left bronchus, 2 months	None	7 mm.	Large many- pointed object im- pacted below a centric stricture	Cupped	Globular end of one spike	Extraction Ultimate cure	30 min. 3 sec.	Oral	<p>First esophagoscopy: The jackstone is very tightly impacted apparently in the lower third of the esophageal wall. Only gentle manipulations were attempted and these demonstrated that there was resistance downward as well as upward. There is an ulceration of the esophageal wall about 4 cm. above the present location of the jack. At this point a large flap of mucosa-covered esophageal wall projects out into the lumen of the esophagus, forming a polypoid mass over the raw surface, which is downward. Jackstone is surrounded by fungations and shreds of lacerated tissue.</p> <p>Second esophagoscopy: Stricture observed at crossing of left bronchus. Lobule of tissue projecting into lumen from the right. Stricture dilated. Foreign body gradually moved upward past one observation after another. Esophagus healed and shows no tendency to heal since last inspected. Subsequent history: Atresia of esophagus followed. Gastrostomy done. Lumen restored and perfect esophageal reaction with complete cure. Retrograde esophagoscopy followed by retrograde bougienage.</p>





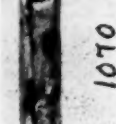
Fbdy. 926  926	1 yr.	Toy horse	Esophagus below cricopharyngeus, 18 days	None	7 mm.	Anci- red by tail and hind leg	Side- curved	Tail and hind leg, alter- nately	Extraction Cure	6 min. 10 sec.	Oral	Removed by esophago- scopic version.
Fbdy. 945  945	5 yrs.	Portion of foot of rub- ber doll	Right main bron- chus, 15 days	None	5 mm.	Impending asphyxia	Side- curved		Extraction Cure	2 min. 46 sec.	Oral	After disimpaction from right main bronchus rub- ber turned crosswise at bifurcation; plus the pus it occluded the left bron- chus and the right bronchus became unusu- ally small because of the pa- thology of many weeks; duration the child ceased breathing for a period of about one minute before the body could be re- stored by tilting the foreign body with the side-curved forceps and passed down into the left lung (the left lung being the unimpaired one).
Fbdy. 947  947	4 yrs.	Toy plate	Esophagus just below crico- pharyngeus, 6 days	None	Esoph- ageal specu- ulum	Large diak with thin rough edges	Down- jaw alliga- tor	Flatwise	Extraction Cure	50 sec.	Oral	Folds of esophageal wall were manipulated away from rough edges of fbdy. with lip of esophageal speculum.

## TOYS—Continued

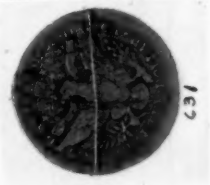

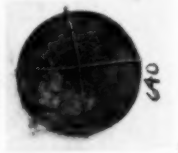
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
	3 yrs.	Piece of crayon	Trachea, 16 days	None	5 mm.	Soft, slippery, waxy nature of body. Lacking elasticity (softened by body temperature) and tendency to go into lower lobe bronchus because of greater air blast into the upper lobe; danger of pushing fragment into lower lobe bronchus	Fenestrated peanut		Extraction Cure	3 min.	Oral	Tracheotomy required before bronchoscopy to prevent drowning of patient in its purulent secretion. The foreign body was removed at subsequent (oral) bronchoscopy, 8 min. 15 sec. Further x-ray study showed position of body; remaining. Second oral bronchoscopy. Mass of crayon found in bifurcation of lower lobe bronchus; left upper lobe bronchus apparently clear of obstruction; on loosening crayon it became fragmentary and secretions were sucked through the crayon. One day later two other portions of crayon were expelled through tracheotomy cannula.
	3 yrs.	Toy jack	Esophagus below cricopharyngeus, 21 days	None	7 mm.	Withdrawal of a flange with six prongs through the folds of the cervical esophagus and into the cricopharyngeus	Ball		Extraction Cure	3 min. 45 sec.	Oral	Fbody, tightly anchored by the second flange anteriorly and the pharyngeal muscle posteriorly. Sized with hollow forceps so as to permit some degree of wabbling and thus of working upward through the stricture. The flange was manipulated out of the way with the tube-mouth.

Fbdy. 997	15 mos.	Toy jack	Esophagus, 23 days	None	7 mm.	Withdrawal of a 6-pronged object, which swollen and lacerated folds	Cupped	One of the knobs	Extraction 9 min. 25 sec.	Oral	Jacksone found buried in stomach filled with food granulation tissue posteriorly and slightly to the right of the true axis of the oesophagus.
Fbdy. 999	3 yrs.	Whistle	Esophagus, 38 days	None	7 mm.	Prevention of food and water while starving for subsistence of the acute traumatic esophageal stenosis	Plain	Flatwise	Extraction 2 min. 21 sec.	Oral	Child was admitted with spontaneous emphysema, cervical cellulitis, drooling, inability to swallow. After a drop of water, water was constantly retained. A constant colonic distention for 7 days, when ability to swallow liquids was slowly regained. No esophagoscopy done by us until 4 weeks after admission, by which time the child could swallow soft solids. Then esophagoscopy was done and whistle removed. It was found buried in posterior wall with an accumulation of mucus and food above. No signs of inflammation. No signs of healed trauma visible. Cicatrices visible above cricopharyngeal fold on posterior wall. No granulation tissue.
Fbdy. 1037	3 yrs.	Small Christmas tree ornament	Left bronchus between bifurcation and orifice of upper lobe bronchus, 5 days	None	5 mm.	Very friable foreign body	Plain	Flatwise in fragments	Extraction 4 min. 13 sec.	Oral	

## TOYS—Continued




Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1046  1046	6 yrs.	Jackstone	Esophagus, 5 days	None	7 mm.	Prongs barbed in incised folds	Ball bearing	By one of ball impaled ends	Extraction Death	10 min. 10 sec.	Oral	Jackstone found imbedded in folds below crico- pharynx. Difficult of removal increased very much by dyspnea. One of pointed branches of jack continually present- ing and version was re- quired to bring up one of the ball-shaped ends. Death occurred after re- moval from abscess of mediastinum following cellulitis of neck.
Fbdy. 1059  1059	4 yrs.	Whistle	Esophagus at suprasternal notch, 12 days	None	Ante- rior com- mis- sure laryn- go- scope	Presenta- tion	Mosher	Flatwise	Portion ex- tracted; portion passed by by bowel Cure	1 min. 10 sec.	Oral	
Fbdy. 1070  1070	5 yrs.	Brass reed from horn	Left bronchus 2 cm. below bi- furcation, 6 days	None	6 mm.	Tight im- paction; mucous secretions in over- edge of foreign body all around its per- iphery	Mos- quito	Edge, one blade in- cised other blade outside	Extraction Cure	11 min. 20 sec.	Oral	

COINS AND OTHER DISKS

Fbdy. 631 	3 yrs.	Coin (Quarter)	Esophagus below cricopharyngeus, 2 days	None	7 mm.	Presenta- tion	Plain	Flatwise	Extraction Cure	1 min.	Oral	
Fbdy. 632 	16 mos.	Coin (Penny)	Larynx, 2 days	None	7 mm.	Avoidance of false passages	Side- curved	Flatwise	Extraction Cure	6 min. 4 sec.	Oral	Esophagus lacerated. Long shreds of mucosa resemb- ling worms. Coin found in larynx. Removed with esophageal forceps after exploring esoph- agus.
Fbdy. 640 	2 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 8 days	None	7 mm.	None	Side- curved	Flatwise	Extraction Cure	28 sec.	Oral	Severe esophagitis.

## COINS AND OTHER DISKS—Continued



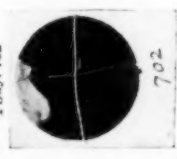
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 642 	2½ yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 3 mos., 13 days	None	7 mm.	Presenta- tion	Side- curved	Flatwise	Extraction Cure	19 sec.	Oral	Ulceration and edema of esophagus.
Fbdy. 649 	2 yrs.	Brass disk (Medal)	Esophagus; upper third, 9 days	None	7 mm.	Tight in- sertion esophagi- tis	Plain	Flatwise	Extraction Cure	41 sec.	Oral	Severe esophagitis. Mucosa excoriated at right and left edges. Mucosa inflamed anteriorly and posteriorly.
Fbdy. 655 	1 yr. 7 mos.	Coin (Penny)	Esophagus below cricopharyngeus, 8 days	None	7 mm.	None	Side- curved	Flatwise	Extraction Cure	10 sec.	Oral	Localized esophagitis. Mucopus surrounding coin.

Fbdy. 656		18 yrs.	Coin (Half dollar)	Esophagus below cricopharyngeus, 16 hours	None	9 mm.	None	Side-curved	Flatwise	Extraction 48 sec. Cure	Oral	Localized esophagitis.
Fbdy. 657		2 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 8 days	None	7 mm.	Presentation	Side-curved	Flatwise	Extraction 3 min. 7 sec. Cure	Oral	Edematous esophagitis, above penny.
Fbdy. 665		1 1/2 yrs.	Coin (Nickel)	Esophagus below cricopharyngeus, 4 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction 1 min. 17 sec. Cure	Oral	Localized esophagitis. Coin embedded in folds of esophagus.

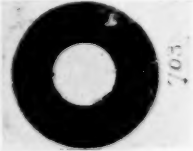
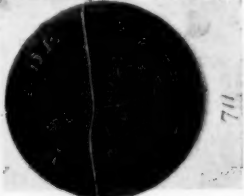
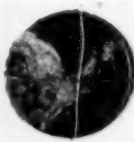
## COINS AND OTHER DISKS—Continued



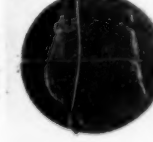
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 671 	21 mos.	Coin (Nickel)	Esophagus below cricopharyngeus, 16 days	None		Presentation	Side-curved	Flatwise	Extraction Cure	10 sec.	Oral	Practically no local reaction.
Fbdy. 680 	3 yrs.	Coin (Fenny)	Esophagus below cricopharyngeus, 2 days	None		Presentation	Plain	Flatwise	Extraction Cure	2 min. 31 sec.	Oral	Localized esophagitis. Small whitish threads in both pyriform sinuses.
Fbdy. 681 	11 mos.	Coin (Fenny)	Esophagus below cricopharyngeus, 8 days	None		Presentation	Plain	Flatwise	Extraction Cure	21 sec.	Oral	





 <p>Fbdy. 691</p>	2 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 6 days	None	7 mm.	Coin buried in esopha- geal folds	Plain	Flatwise	Extraction Cure	4 min. 48 sec.	Oral	Edematous esophagitis. Coin buried in folds and covered with decom- posed solid food.
 <p>Fbdy. 692</p>	1 yr.	Coin (Penny)	Esophagus below cricopharyngeus, 2 months	None	Laryn- go- scope	Presenta- tion	Plain	Flatwise	Extraction Cure	1 min. 2 sec.	Oral	Folds swollen. Dyspnea during esophagoscopy.
 <p>Fbdy. 702</p>	2 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 6 days	None	7 mm.	Presenta- tion	Plain	Flatwise	Extraction Cure	4 min. 42 sec.	Oral	Accumulation of food and gelatinous material above coin.




## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 703 	3 yrs.	Metal washer	Esophagus below cricopharyngeal fold	None	7 mm.	None	Side- curved	Flatwise	Extraction Cure	35 sec.	Oral	Slight localized esophagitis.
Fbdy. 711 	10 yrs.	Brass check	Esophagus below cricopharyngeus, 5 days	None	7 mm.	Presenta- tion	Side- curved	Flatwise	Extraction Cure	2 min. 10 sec.	Oral	Foreign body tightly clamped by esophageal walls.
Fbdy. 730 	3 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 1 week	None	Anter- ior con- densed sure laryn- go- scope	Presenta- tion	Plain	Flatwise	Extraction Cure	1 min. 44 sec.	Oral	

<p>Fbdy. 737</p>  <p>737</p>	<p>3 yrs.</p>	<p>Coin (Nickel)</p>	<p>Esophagus below cricopharyngeus, 2 days</p>	<p>None</p>	<p>Esoph- ageal spec- ulum</p>	<p>Avoidance of lesions</p>	<p>Plain</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>47 sec.</p>	<p>Oral</p>	<p>Intense esophagitis with numerous abrasions.</p>
<p>Fbdy. 738</p>  <p>738</p>	<p>18 mos.</p>	<p>Coin (Penny)</p>	<p>Esophagus below cricopharyngeus, 4 months</p>	<p>None</p>	<p>Esoph- ageal spec- ulum</p>	<p>Avoidance of false passages</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>16 min. 42 sec.</p>	<p>Oral</p>	<p>Fistula in anterior wall of esophagus. Looked dan- gerous to enter with esophagoscope. Lips were spread with esophageal speculum when speculum of coin was introduced. False passage was seen to be split out at the side; it was not a blind false passage.</p>
<p>Fbdy. 740</p>  <p>740</p>	<p>3 yrs.</p>	<p>Coin (Nickel)</p>	<p>Esophagus at crossing of left bronchus, 3 mos.</p>	<p>None</p>	<p>7 mm.</p>	<p>Presenta- tion</p>	<p>Plain</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>1 min. 30 sec.</p>	<p>Oral</p>	<p>Coin found in lumen of esophagus covered with detritus and exudate at level of crossing of left bronchus. No sign of traumatism; slight esoph- agitis just above coin.</p>




## COINS AND OTHER DISKS—Continued



Case Number and Illustration	Age	Foreign Body	Location	Anes-thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Body. 744  744	3 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 4 days	None	7 mm.	Presentation	Side-curved	Flatwise	Extraction Cure	2 min. 48 sec.	Oral	Penny covered with food and lodged tightly against posterior wall just below cricopharyngeus.
Body. 745  745	5 yrs.	Coin (Nickel)	Esophagus below cricopharyngeus, 5 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure	19 sec.	Oral	

Fbdy. 75A		2 yrs.	Coins (Two pennies)	Esophagus below cricopharyngeal fold, 17 days	None	7 mm.	None	Plain	Flatwise (both coins as one)	Extraction Cure	16 sec.	Oral	Pharynx bleeding freely. Source of blood not determined except one large surface covered with granulations on the posterior wall.
Fbdy. 762		7 yrs.	Coin (Quarter)	Esophagus below cricopharyngeum, 5 days	None	7 mm.	None	Side-curved	Flatwise	Extraction Cure	44 sec.	Oral	
Fbdy. 771		3 yrs.	Coin (Nickel)	Esophagus below cricopharyngeum, 17 days	None	7 mm.	Swollen folds	Side-curved	Flatwise	Extraction Cure	59 sec.	Oral	

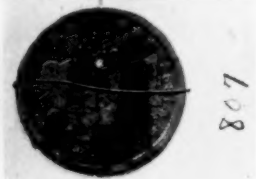

Coin very much corroded; corrosion being of a dark straw color.

## COINS AND OTHER DISKS—Continued


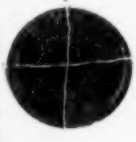
Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 780  780	5 yrs.	Tin disc	Esophagus at junction of left bronchus, about 6 months	None	7 mm.	Ulceration, granular, swollen folds	Plain	Flatwise	Extraction Cure	1 min. 48 sec.	Oral	Ulceration at both edges where disc was enclosed in anteroposteriorly closing folds on both sides.
Fbdy. 791  791	3 yrs.	Coin (Nickel)	Esophagus just below cricopharynx, under the overhanging of cricopharyngeal fold	None	7 mm.	None	Plain	Flatwise	Extraction Cure	35 sec.	Oral	
Fbdy. 796  796	21 yrs.	Coin (Quarter)	Esophagus just below cricopharyngeal fold	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure	2 min. 41 sec.	Oral	Esophagus showed no signs of irritation or reaction.

Fbdy. 797		2½ yrs.	Coin (Penny)	Esophagus below cricopharyngeal fold, 21 days	None	7 mm.	Avoidance of false passages already existing	Plain	Flatwise	Extraction Cure	Oral	<p><i>First esophagoscopy.</i> Large perforation of the esophagus at the level of the upper border of the cricoid cartilage. Pocket looks to be about 2 cm. deep. Sloughs to right of upper edge of pocket. Esophagoscopy not deemed advisable at present. Advise patient and calomel to be given dry on tongue for a few days. Time: 2 min. 28 sec.</p> <p><i>Second.</i> Esophagus sloughing. False passage not made. The edges of the laryngopharyngeal walls still gaping. The edges could not be drawn together sufficiently to make it safe to pass the esophagoscopic tube. Time: 3 min. 21 sec.</p> <p><i>Third.</i> Difficulty. Extremely firm pressure on the anterior wall with the esophagoscopic tube. Unable to pass. To avoid entering false passage. This anterior pressure interrupted breathing and could only be carried out intermittently in order to give child air between attempts. Child removed tube. Time: 4 min. 21 sec.</p>
Fbdy. 803		16 mos.	Coin (Penny)	Esophagus below cricopharyngeus, 11 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction 1 min. 49 sec. Cure	Oral	

## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 807 	3½ yrs.	Coin (Quarter)	Esophagus just below crico- pharyngeal fold, 4 days	None	7 mm.	Coin very firmly an- chored be- low crico- pharyn- geus	Side- curved	Flatwise	Extraction Cure	2 min.	Oral	Coin found embedded be- low the lower border of cricoid cartilage, firmly anchored. Strong trac- tion was avoided. Coin freed with lip of esophag- oscope and it came away readily on very gentle traction.
Fbdy. 810 	2 yrs. 7 mos.	Coin (Penny)	Esophagus below cricopharyngeal fold, 1 week	None	7 mm.	Impaction; swollen folds	Side- curved	Flatwise	Extraction Cure	1 min.	Oral	Coin found jammed below cricoid, forced later- ward and behind and lib- erated by contractions of the cricopharyngeus.





 Fbdy. 816	13 yrs.	Coin (Half dollar)	Esophagus just below cricopharyngeal fold, 1 day	None	7 mm.	Presen- tation	Plain	Flatwise	Extraction Cure	1 min. 12 sec.	Oral	<p>1st Inspection esophagus: Hypopharynx in shreds; ulcerations in a number of different locations. Large slit-like perforation on posterior wall. Lifting larynx forward so many sloughs appeared that it was decided best to postpone removal of the coin until esophagitis subsides and adhesions protect the false passages. 2nd Inspection: Posterior esophageal wall sloughing in two large patches, 1 min. 25 sec. Esophagocopy. Granulations still visible, the patch being about one cm. by two cm. in size. Penny found in esophagus back of heart below crossing of left bronchus. Fbdy. removed, 2 min. 10 sec.</p>
 Fbdy. 829	2 yrs.	Coin (Penny)	Esophagus below crossing of left bronchus, 30 days	None	7 mm.	Avoidance of existing false passages	Side-curved	Flatwise	Extraction Cure		Oral	




816

829




155




## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 830 	2 yrs. 9 mos.	Coin (Quarter)	Esophagus below cricopharyngeal fold, 5 days	None	7 mm.	Obscured by food	Side- curved	Flatwise	Extraction Cure	1 min. 2 sec.	Oral	Foreign body buried in mass of food.
Fbdy. 842 	3 yrs.	Coin (Quarter)	Esophagus below cricopharyngeal fold	None	7 mm.	Presenta- tion	Side- curved	Flatwise	Extraction Cure	1 min. 7 sec.	Oral	No local reaction.



Fbdy. 864  864	4 yrs.	Coin (Nickel)	Esophagus below cricopharyngeus, 9 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure	1 min. 2 sec.	Oral	Coin stained straw color all over.
Fbdy. 866  866	9 yrs.	Coin (Quarter)	Esophagus below cricopharyngeal fold, 2 weeks	None	7 mm.	Coin im- pacted deeply in fold of inflamma- tory tissue	Side- curved	Flatwise	Extraction Cure	2 min.	Oral	
Fbdy. 868  868	2 yrs.	Coin (Nickel)	Esophagus below cricopharyngeus	None	7 mm.	None	Plain	Flatwise	Extraction Cure	40 sec.	Oral	

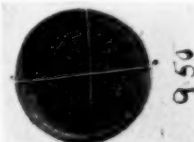
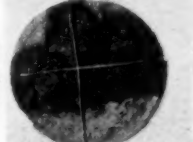
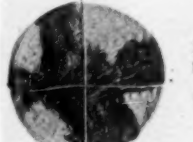
## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 878 	5 1/2 yrs.	Coin (Quarter)	Esophagus about 3 cm. below crico- pharyngeal fold, 5 days	None	7 mm.		Plain	Flatwise	Extraction Cure	40 sec.	Oral	
Fbdy. 880 	4 yrs.	Coin (Quarter)	Esophagus below cricopharyngeal fold, 2 days	None	7 mm.	Obscured by food	Side- curved	Flatwise	Extraction Cure	4 min. 17 sec.	Oral	Quarter very much ob- scured by mass of food impacted above it.
Fbdy. 896 	2 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 10 days	None	7 mm.	Presenta- tion	Alligator	Flatwise	Extraction Cure	1 min. 4 sec.	Oral	



<p>Fbdy. 906</p>  <p>906</p>	<p>2 yrs.</p>	<p>Coin (Penny)</p>	<p>Esophagus below cricopharyngeal fold, 1 week</p>	<p>None</p>	<p>7 mm.</p>	<p>Covered by swollen mucosal folds</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>2 min. 32 sec.</p>	<p>Oral</p>	<p>First esophagoscopy for inspection of the esophagus and extractions of esophagus extending from the cricoid down to the crossing of the left bronchus; coin not seen in any of these lacerated areas. Second esophagoscopy revealed a foreign body embedded in swollen mucosa on posterior wall of esophagus below cricopharyngeus; coin surrounded by large area of esophagitis. Removed.</p>
<p>Fbdy. 909</p>  <p>909</p>	<p>5 yrs.</p>	<p>Coin (Quarter)</p>	<p>Esophagus below cricopharyngeus</p>	<p>None</p>	<p>7 mm.</p>	<p>Avoidance of false passages and traumatic lesions</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>2 min. 35 sec.</p>	<p>Oral</p>	<p>First esophagoscopy for inspection of the esophagus and extractions of esophagus extending from the cricoid down to the crossing of the left bronchus; coin not seen in any of these lacerated areas. Second esophagoscopy revealed a foreign body embedded in swollen mucosa on posterior wall of esophagus below cricopharyngeus; coin surrounded by large area of esophagitis. Removed.</p>
<p>Fbdy. 916</p>  <p>916</p>	<p>6 yrs.</p>	<p>P. R. R. brass disc</p>	<p>Esophagus below cricopharyngeal fold, 7 days</p>	<p>None</p>	<p>Esophagical ulcerum</p>	<p>Presenta- tion</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>1 min. 10 sec.</p>	<p>Oral</p>	<p>First esophagoscopy for inspection of the esophagus and extractions of esophagus extending from the cricoid down to the crossing of the left bronchus; coin not seen in any of these lacerated areas. Second esophagoscopy revealed a foreign body embedded in swollen mucosa on posterior wall of esophagus below cricopharyngeus; coin surrounded by large area of esophagitis. Removed.</p>

## COINS AND OTHER DISKS—Continued



Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 922 	3 yrs.	Tin whistle	Esophagus below cricopharyngeal fold, 7 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure		Oral	
Fbdy. 935 	2 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 11 days	None	7 mm.	Obscured by food and hair	Side- curved	Flatwise	Extraction Cure	1 min. 22 sec.	Oral	Coin found looking like a greyish mass, thickened, rounded and of irregular shape. It was surrounded by a mass that surrounded the coin was discovered to be hair.

<p>Fbdy. 950</p> 	<p>3 yrs.</p>	<p>Coin (Nickel)</p>	<p>Esophagus below cricopharyngeus muscle, 4 days</p>	<p>None</p>	<p>7 mm.</p>	<p>None</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction 35 sec. Cure</p>	<p>Oral</p>	
<p>Fbdy. 952</p> 	<p>3 yrs.</p>	<p>Coin (Quarter)</p>	<p>Esophagus below cricopharyngeus, 13 days</p>	<p>None</p>	<p>7 mm.</p>	<p>None</p>	<p>Side- curved</p>	<p>Flatwise</p>	<p>Extraction 30 sec. Cure</p>	<p>Oral</p>	<p>Localized esophagitis; fermented food adhering to coin giving it a foul odor. Coin somewhat corroded.</p>
<p>Fbdy. 957</p> 	<p>15 mos</p>	<p>Coin (Quarter)</p>	<p>Esophagus just below crico- pharyngeal fold, 4 days</p>	<p>None</p>	<p>7 mm.</p>	<p>None</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction 24 sec. Cure</p>	<p>Oral</p>	<p>Coin had a cross stained on it from sulphides. Evidently after being stained in one diameter it had turned nearly 90 degrees and a cross was formed in another diameter.</p>


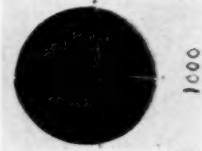
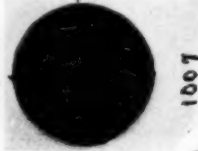
## COINS AND OTHER DISKS—Continued



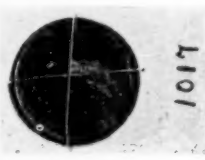
Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Problem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 959 	15 mos.	Coin (Penny)	Esophagus below cricopharyngeus, 12 days	None	Laryn- scope	Avoidance of lesions	Alligator	Flatwise	Extraction Cure	3 min.	Oral	Intense esophagitis. Slough covering granulating area on posterior wall.
Fbdy. 960 	8 yrs.	Coin (Nickel)	Esophagus below cricopharyngeus 2 days	None	7 mm.	None	Side- curved	Flatwise	Extraction Cure	50 sec.	Oral	
Fbdy. 961 	16 mos.	Coin (Penny)	Esophagus below cricopharyngeus, 4 days	None	7 mm.	None	Alligator	Flatwise	Extraction Cure	50 sec.	Oral	





<p>Fbdy. 972</p> 	11 yrs.	Coin (Half dollar)	Esophagus 30 cm. from upper teeth, 1 day	None	7 mm.	Disimpaction and manipulation of obstructing folds during withdrawal	Side-curved	Flatwise	Extraction Cure	3 min.	Oral	
<p>No. 977 No specimen</p>	18 mos.	Coin (Penny)	Esophagus at hiatus esophageus 4 days	None	7 mm.		Plain		Coin escaped downward into stomach; passed	5 min. 55 sec.		Fbdy. doubtless escaped downward during gagging at start of esophagoscopy.
<p>Fbdy. 993</p> 	2 yrs.	Coins (2 pennies)	Esophagus at hiatus esophageus, 1 week	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure	1 min. 13 sec.	Oral	Two pennies found stuck together, lodged about 1 cm. above hiatal opening. So far as could be determined hiatal opening did not seem smaller or more tightly contracted than normal.




## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 998 	2 yrs. 5 mos.	Coin (Penny)	Esophagus below cricopharyngeus, 3 days	None	7 mm.	Presenta- tion	Plain	Flatwise	Extraction Cure	3 min. 1 sec.	Oral	
Fbdy. 1000 	2 yrs.	Metal "slug"	Esophagus below cricopharyngeus, 6 days	None	7 mm.	Presenta- tion	Side- curved	Flatwise	Extraction Cure	1 min. 50 sec.	Oral	No irritation of surround- ing mucosa.
Fbdy. 1007 	2 yrs.	Coin (Nickel)	Esophagus 1 cm. below crico- pharyngeus, 4 days	None	7 mm.	Presenta- tion	Side- curved	Flatwise	Extraction Cure	1 min. 4 sec.	Oral	

<p>Fbdy. 1015</p> 	<p>2½ yrs.</p>	<p>Coin (Nickel)</p>	<p>Esophagus below cricopharyngeus, 7 days</p>	<p>None</p>	<p>Ante- rior com- mis- sure laryn- goscope</p>	<p>Presenta- tion</p>	<p>For- ward grasp- ing</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>2 min. 11 sec.</p>	<p>Oral</p>	
<p>Fbdy. 1016</p> 	<p>2 yrs.</p>	<p>Coin (Penny)</p>	<p>Esophagus at suprasternal notch, 1 day</p>	<p>None</p>	<p>Ante- rior com- mis- sure laryn- goscope</p>	<p>Presenta- tion</p>	<p>Alligator</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>3 min. 30 sec.</p>	<p>Oral</p>	
<p>Fbdy. 1017</p> 	<p>10 mo.</p>	<p>Coin (penny)</p>	<p>Esophagus below 3 tracheal rings, 3 weeks</p>	<p>None</p>	<p>7 mm.</p>	<p>Foreign body buried in esophageal folds ob- scured by white pasty material</p>	<p>For- ward and grasp- ing</p>	<p>Flatwise</p>	<p>Extraction Cure</p>	<p>1 min. 25 sec.</p>	<p>Oral</p>	




## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anes- thetic	Tube	Prob- lem	For- ceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1027 	13 yrs.	Metal disc Dredged from lead ladle	Esophagus below cricopharyngeus, 16 days	None	8 mm.	Tightly buried in esophageal folds Enormous size and heavy weight	Plain	Flatwise	Extraction Cure	3 min. 25 sec.	Oral	
Fbdy. 1028 	4 1/2 yrs	Coin (quarter)	Esophagus, 1 day	None	Ante- rior com- mis- sure laryn- go- scope	Presenta- tion	Alligator	Flatwise	Extraction Cure	2 min. 20 sec	Oral	




Fbdy. 1029		3 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 1 day	None		Presenta- tion	Plain	Flatwise	Extraction Cure	1 min. 35 sec.	Oral	
Fbdy. 1033		6 yrs.	Coin (Penny)	Esophagus at crossing of left bronchus, 2 days	None			Plain	Flatwise	Extraction Cure	3 min. 35 sec.	Oral	
Fbdy. 1035		3 yrs.	Coin (Nickle)	Esophagus just below crico- pharyngeus, 9 days	None	7 mm.	Presenta- tion free of folds	Plain	Flatwise	Extraction Cure	1 min. 9 sec.	Oral	Yellow discoloration on coin.

## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Seizure	Result	Time	Route	Comment
Fbdy. 1047 	21 mos.	Coin (Penny)	Esophagus at crossing of left bronchus, 7 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure	2 min. 53 sec.	Oral	Coin darkly corroded, looking brown on presentation.
Fbdy. 1050 	3 yrs.	Coin (Nickle)	Esophagus below cricopharyngeal fold, 7 days	None	Anterior commissure laryngoscope	Presentation	Alligator	Flatwise	Extraction Cure	2 min. 42 sec.	Oral	Yellow discoloration.
Fbdy. 1053 	3 yrs.	Coin (Penny)	Esophagus below cricopharyngeal, 2 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure		Oral	Coin darkly discolored.

Fbdy. 1063  1063	13 mos.	Coin (Penny)	Esophagus, 3 days	None	Child's laryn- go- scope	Presenta- tion	Alligator (Mosh- er's)	Flatwise	Extraction Cure	Oral	Darkly discolored on cen- tral zone.
Fbdy. 1065  1065	2½ yrs.	Coin (Penny)	Esophagus just below crico- pharyngeus, 14 days	None	7 mm.	Presenta- tion	Side- curved	Flatwise	Extraction Cure	Oral	Dark streak of discolora- tion noted on central zone of coin. Particles of food adherent.
Fbdy. 1089  1089	2 yrs.	Coin (quarter)	Esophagus just back of heart, 11 days	None	7 mm.	None	Plain	Flatwise	Extraction Cure	Oral	Central zone discolored.

## COINS AND OTHER DISKS—Continued

Case Number and Illustration	Age	Foreign Body	Location	Anesthetic	Tube	Problem	Forceps	Point of Suture	Result	Time	Route	Comment
Fbdy. 1102  1102	3 yrs.	Coin (Penny)	Esophagus below cricopharyngeus, 25 days	None	7 mm.	Presentation	Plain	Flatwise	Extraction Cure	2 min. 25 sec.	Oral	
Fbdy. 1137  1137	3 yrs.	Disc	Esophagus below cricopharyngeus, 5 days	None	Anterior commissure laryngoscope	Presentation	Rotation	Flatwise	Extraction Cure	2 min. 24 sec.	Oral	
Fbdy. 1149  1149	4 yrs.	Coin (Quarter)	Esophagus	None	7 mm. retrograde	Presentation	Side-curved	Flatwise	Extraction Cure	2 min. 47 sec.	Oral	



<p>Bronchoscopy (oral) revealed a somewhat irregular patch of membrane on the intensely swollen mucous membrane projecting in from the left. Trachea intensely inflammatory. Just below the tracheostomic incision the trachea was found to be completely plugged with a rounded mass of false membrane which looked just like nasal polyp. On removing this plug two other plugs were seen, the one in the right and the other in the left bronchus. On removal of these adherent patches of membrane were seen lower down. These deeper membranes were not readily detached. Antiseptic applied. Many similar cases omitted from these tables.</p>																				
<p>No. 849 No specimen</p>	<p>5 yrs.</p>	<p>Diphtheritic membrane</p>	<p>Trachea and bronch</p>	<p>None</p>	<p>5 mm.</p>	<p>Impending asphyxia</p>	<p>Side-ward; also aspirating tube</p>	<p>By pre-arranging part</p>	<p>Extraction Death on way to Municipal Hospital</p>	<p>18 min. 2 sec.</p>	<p>Oral</p>									
<p>Fbdy. 000</p>	<p>20 yrs.</p>	<p>Broncholith; siliceous granules</p>	<p>Left lower lobe</p>	<p>Local</p>	<p>7 mm. aspirating</p>	<p>None</p>	<p>Sponge carrier with gauze</p>		<p>Removal; suppuration unimproved</p>	<p>19 min. 42 sec.</p>	<p>Oral</p>									<p>Long-suppression of long standing. Bronchoscopy done for diagnosis. Previous diagnosis of bronchiectasis confirmed by this bronchoscopy. Transposition of the lungs.</p>



LXIX.

CICATRICAL STENOSIS OF THE ESOPHAGUS,  
WITH PARTICULAR REFERENCE TO TREAT-  
MENT BY CONTINUOUS STRING, RETRO-  
GRADE BOUGINAGE WITH THE  
AUTHOR'S BOUGIE.\*

By GABRIEL TUCKER, M. D.,

PHILADELPHIA.

Etiology of Stenosis.—In cases of cicatricial stenosis coming to the Bronchoscopic Clinic of Dr. Chevalier Jackson, lye burns have been by far the most common cause of stricture. Guisez, in his statistics of 2,500 cases of esophageal disease which he examined esophagoscopically, reports one in twelve, or 185 cases, to be cicatricial stenosis. All of these 185 patients had swallowed caustics, except seven whose stenosis was due to foreign body; two from burns of hot food, five from injuries from war projectiles, and two from swallowing toxic gas.<sup>7</sup> Zimanyi states that in the last three years, 10 per cent of the patients in the nose and throat departments of the Budapest and Berlin hospitals have been cases of stenosis of the esophagus from caustic burns from lye solution swallowed in mistake for drinking water, the people of Hungary and Germany using the lye solution for home soapmaking.

Another cause of cicatricial stenosis is burn from acids. bichlorid of mercury, ammonia, sulphuric acid, etc. Systemic diseases, such as typhoid fever, diphtheria, tuberculosis, lues, scarlet fever, and various pyogenic conditions, can produce ulceration followed by cicatrices of the esophagus. At the present time we have under treatment in the Bronchoscopic Clinic three cases of cicatricial stenosis following typhoid fever, three young males, all coming from the same community in central West Virginia. Stupka reports nine undoubted

\*Accepted candidate's thesis read before the meeting of the American Laryngological, Rhinological and Otological Society, May 31, 1924.

cases of cicatricial stenosis caused by diphtheria.<sup>24</sup> This disease would probably be an even more frequent causative factor in cicatricial stenosis, were it not that in severe cases of diphtheria the patient dies of the systemic disease before the cicatrices form in the ulcerated esophagus. DaCosta states that 15 per cent of the cases of scarlet fever may result in cicatricial stenosis.<sup>2</sup> Observations by Guisez, Jackson, MacKinney prove that organic stricture may result from chronic esophagitis, secondary to spasmodic lesions. The prolonged sojourn of a foreign body, where inflammatory infiltration results from a long continued ulcerative process, may cause stricture. War wounds and gunshot wounds and the wounds of unskillful attempts at removal of a foreign body may result in obstructive cicatrix. A case of this kind came to the Bronchoscopic Clinic and is included in the accompanying reports of cases. Occasionally a burn from hot food or beverage causes formation of scar tissue, and Gottstein reports one case resulting from the swallowing of hot potato which the patient was unable to reject.<sup>7</sup> Peptic ulcer at the lower end of the esophagus may be a cause, as Jackson points out.<sup>12</sup>

**Age of Patients.**—Of the cases of cicatricial stenosis of the esophagus who have come to the Bronchoscopic Clinic, by far the greatest number are children, chiefly cases of lye burn. The remaining relatively small percentage is adults, some of whom have taken poison accidentally or with suicidal intent; others have come with lesions from foreign body; still others have a cicatrix as a sequel to systemic disease. This latter type of stenosis rarely occurs in children. Guisez reports 98 children to 87 adults; and von Acker's experience has been that out of 100 cases 44 were children, 50 were adults, and in six the age was not recorded.<sup>12</sup>

**Type of Stricture.**—The stricture may be single, at one level. This type, however, is more frequently congenital than cicatricial. In cicatricial stenosis the strictures are usually multiple at two or more levels, particularly after caustic burns, and are more frequently eccentric than annular.

**Location of Stricture.**—Strictures as the result of corrosion from caustics are found most often at the level of the anatomic or physiologic narrowings of the esophagus. According to Chevalier Jackson,<sup>12</sup> they occur first, in point of frequency,

at the level of the crossing of the left bronchus, next in the region of the cricopharyngeus, next at the hiatal level. Stricture at the cardia is rare. Mosher<sup>19</sup> mentions the frequency of cicatricial stricture at the beginning of the liver tunnel, due to the delay of hot food and caustics at this point, and to the tendency of the "hammering" of the liver "to reinforce ulcerative processes and to localize strictures at this level."

**Esophagosopic Appearance.**—The approach of the esophagoscope through the strictured area frequently shows the esophagus to be dilated, with a pouch deeper on one side than the other. The strictured area is somewhat paler than the surrounding mucosa, the scar itself usually being pale or white. It may entirely surround the lumen, but more frequently the scar tissue involves only a part of the circumference of the walls. The scar tissue may form a hard ridge, or be flat and level with the mucosa, or even be indented. The mucosa of the dilated area may be inflammatory, and granulations and ulcerations may be present, especially if the case presents itself after recent injury. If the stricture is multiple, there is dilatation between the constrictions, with retention of food and secretions. It is necessary, of course, that the esophagus be cleansed of food débris and retained secretions, in order to be sure of conditions present. Lavage for the removal of the food débris, and the administration of bismuth subnitrat with the addition of a little calomel dry on the tongue, is effective in allaying the inflammatory process.

**Prognosis.**—The mortality of untreated strictures of the esophagus is very high. Clearly defined stricture, left to itself, is eventually fatal. Strictures from caustic burns are more serious than those from other causes, because the stenosis is more severe. Before the days of esophagoscopy, in statistics based on a large number of cases, von Acker gave a mortality of 40 to 50 per cent. In Billroth's series of cases, from 1877 to 1886, a mortality of 38 per cent was given.<sup>7</sup> In general, it may be said that the sooner the case comes under observation and treatment the better the prognosis. Early gastrostomy, the getting of a string through the esophagus before it closes, and very gentle retrograde treatment will ultimately cure practically all of these cases, unless some constitutional effect of the accident arises, such as renal or gastrointestinal lesions. Too

much cannot be said for early gastrostomy in all cases where there is difficulty in swallowing. This is particularly emphasized by Jackson, who states that with the proper tube feeding, life may be prolonged indefinitely. Now, with the use of exceedingly safe and rapid methods of dilatation, the peroral esophagoscopy bouginage of Jackson, or the continuous string retrograde bouginage with the author's bougie, with the possibility of maintaining the gastrostomy opening for emergency, and the continuing of the retrograde treatment by the family physician until the scar tissue has been resorbed, or no longer obstructs, owing to the gradual dilatation of the normal esophageal wall at the stricture level, so that there is no possibility of relapse, we feel that the prognosis is good. The earlier the treatment is begun the less the time required for cure.

Symptoms.—The symptoms that come on immediately after the accident, particularly in lye cases, are due to inability to swallow because of the severe inflammatory reaction, swelling and ulceration. These acute symptoms usually subside at the end of the first week or fortnight, and the swallowing improves and even becomes practically normal for a time. Then gradual dysphagia develops, its rapidity depending upon the extent of the injuries to the esophagus, until there is difficulty, first with solids, later with liquids, and finally there may develop an inability to swallow even saliva. Loss of weight is progressive, owing to the inability of the patient to swallow sufficient food. Dehydration results in severe cases of water starvation. The examination of the pharynx with the laryngeal mirror will show the pyriform sinuses filled with frothy secretion (Jackson's sign of esophageal stenosis). Sometimes aphagia comes on, in severe cases a few weeks after the burn, but occasionally adults appear with history of a burn from caustics many years before. Pain is not usually a symptom.

Diagnosis.—History of burn or trauma is to be considered in making a diagnosis of cicatricial stenosis. Symptoms of dysphagia, with regurgitation and progressive loss of weight, necessitate the exclusion of possible cancer, aneurysm with compression, foreign body and diverticulum before a diagnosis of cicatricial stenosis can be made. The employment of a bougie blindly as a diagnostic means in esophageal obstruction or dysphagia is not justifiable at the present day. It is danger-

ous, inefficacious, and unnecessary since the development of esophagoscopy. A bougie will not distinguish an aneurysm from a stricture, unless perforation of the aneurysm occur. But even esophagoscopic methods should be preceded by roentgen ray examination. Fluoroscopic studies by the swallowing of an opaque mixture, supplemented by film studies, will exclude possible aneurysm, and will often make the differential diagnosis as to other lesions. Dr. Willis F. Manges, by his method of filling the esophagus with an opaque mixture, can distinguish large caliber stricture. Dr. Henry K. Pancoast, by having a soft rubber catheter put through the nasopharynx, was able to inject bismuth mixture into the esophagus of a small child who refused to swallow the mixture, and demonstrated a stricture of very small caliber that could not be otherwise shown. Auscultation and determination of swallowing time is not of great diagnostic value since the development of roentgen ray diagnosis. Esophagoscopic bouginage will facilitate determination of different stricture levels, but should be used with extreme caution. Double, peroral and retrograde esophagoscopic bouginage may be used if there is already a gastrostomic fistula. Lodgment of a bolus of food in a supposedly normal esophagus has been the occasion of a diagnosis of an organic lesion of the esophagus which proved to be a stricture. In the case of a man, sixty years of age, who came to the Bronchoscopic Clinic with a bolus of food lodged in the esophagus, there was found on esophagoscopy a large caliber stricture in the upper third of the esophagus. He was questioned about the possibility of having had a burn of the esophagus and at first denied such a possibility, but later admitted having taken sulphuric acid in his youth, with suicidal intent. This accounted for the stricture which caused the lodgment of the bolus of food.

Methods of Treatment.—The esophagus is one of the most intolerant organs with which one has to deal surgically. Besides being a septic canal which drains all the infectious secretions from the mouth to the stomach, it is susceptible to shock to an extent out of all proportion to the severity of the operation or lesion, as shown in the ordinary acute esophagitis from traumatism. The importance of putting the esophagus at rest, when there is marked narrowing of the canal and in-

ability to swallow sufficient nourishment, is strongly emphasized by Jackson. This is accomplished by early gastrostomy. Mosher also advocates early gastrostomy, particularly in children and emaciated adults.

**Peroral Methods.**—Blind bouginage is mentioned only to be condemned. In 1831 Fletcher stated, "The best of the bougies which act from the point is that made of elastic gum but stiffened with cold, or still farther by freezing. A firm stricture will turn it back and, if stiffened too much, it has all the mischief of the common bougie, for its point must be urged, and if placed wrong it may thus be drawn anywhere but through the stricture."

Trousseau said, "Sooner or later all cases of stricture of the esophagus die of the bougie." Despite these warnings, and similar observations of the ablest esophagoscopists in recent years, we still have occasionally coming to the Bronchoscopic Clinic cases of retropharyngeal abscess and mediastinitis from blind bouginage, and we learn of many fatal cases. Many museums have specimens of perforated esophagus. Esophagoscopic and retrograde bouginage have made this dangerous method obsolete.

Reports of success have been given of the use of flexible necklace-like bougies, made of smooth olivary or rounded bodies strung on fine wire or silk. These are also sometimes used for retrograde bouginage in the seesaw methods. Billroth and others have used conical shaped rubber bougies filled with shot or mercury to stiffen them, but with the same illusory success as the cold stiffened rubber bougie of 1831. The method of Jacobson is probably the most unique and simple method yet devised, although its efficiency is questionable. Relying on the peristalsis and natural swallowing movements, he had the patient swallow a tapered stalk of laminaria with a string attached at the broadened top. He hoped that the tip would engage in the stricture, and noted that the patient complained of a severe substernal pain, and from this concluded that the laminaria was expanding and dilating the stricture as it swelled.<sup>1,2</sup>

By the peroral string-guided methods of Sippy, Mixer and Plummer, the patient swallows a string, which is used as a guide when it is threaded through an eye in the tip of the

bougie. The string is pulled taut and the bougie pushed through the stricture over the string. Ledoux in connection with ballooning method calls attention to the participation of the periesophageal tissues, among others the pulmonary hilum, in the inflammatory processes consecutive to burns. He reports five cases of pleural effusion in the region of work on the stricture.<sup>17</sup>

Forcible divulsion has been occasionally advocated but is now generally abandoned. Divulsion of the upper one of a series of strictures is mentioned by Jackson as a method that the very expert esophagoscopist may use on some occasions, but since the development of my bougie he has ceased to use forcible divulsion, although he still uses esophagoscopic peroral bouginage in cases not gastrotomized.

Internal esophagotomy and surgical reconstruction of the esophagus after cicatricial stricture have been reported as successful by Lotheissen, Narath and others, but each success has been preceded by high mortality.<sup>18</sup> External esophagotomy for cervical strictures is sometimes successful, according to Jackson and DaCosta.

Electrolysis has been used with varying results. Its chief advocate is Guisez, who considers it a most successful method.

Esophagoscopic Bouginage.—The forms of the bougie used through the esophagoscope are various. The olive tipped fili-form bougie has been modified in some instances by the addition of metal or ivory olives of increasing size. Sometimes the steel stem is replaced by a staff of whalebone. Jackson and Guisez give preference to semisoft, flexible, fiber bougies for peroral esophagoscopic bouginage.

Retrograde Bouginage.—The first report of successful retrograde esophagoscopic catheterism was made by Ehrlich in 1897. Previous to this, rapid dilatation by means of rubber capped forceps introduced through the stomach and upward into the stricture had been performed by Loretta, Mickulicz, v. Acker, Jaffe, but the method was attended by high mortality, although success was reported in some cases. The difficulty of finding the hiatus esophageus at retrograde esophagoscopy is emphasized by Jackson, Guisez, and many others, and was the chief objection to retrograde esophagoscopic bou-



ginage before the endless string methods, which necessitated the finding of it only once, were devised.

Billroth introduced the filiform bougie retrograde into the esophagus and attached to it a conical shaped bougie on a string. Abbe in his string sawing method passed a string through and inserted a bougie beside the string tightly into the stricture, and by pulling the string backwards and forwards while an assistant made pressure on the olive of a metallic bougie, thus cut the stricture. Graham<sup>5</sup> modified this method by using the parachute arrangement that enabled him to pull four strings through the strictured area, and these were kept taut in different directions while the bougie was pulled through the lumen opened by the strings. Dunham caused a string to be swallowed and then used the Abbe method. Jackson recommends the Brenneman method of making a lumen in impervious strictures in order to introduce the bougie for retrograde bouginage. By this method an opening is made high up in the stomach, above the gastrostomic fistula, into which the surgeon inserts his finger until it reaches the impervious stricture. He can then feel, on the other side of the stricture, the esophagoscope passed from above, and with comparative safety he can cut the strictured tissue sufficiently to permit a soft rubber tube to be inserted.

Ochsner passes a string from below and pulls through a double rubber tube on the stretch, depending on the resiliency of the rubber tube, when traction is relaxed, for dilatation. Guisez uses the same principle as Ochsner, but pulls in a single rubber tube on the stretch with good results.

Retrograde esophagoscopy, using the steel stemmed filiform bougie of Jackson, is particularly useful to place the string when it cannot be swallowed. Combined retrograde and peroral esophagoscopy has enabled us at the Bronchoscopic Clinic to perforate through the obliterated part of an esophageal atresia to place a string as the first step in a cure. This will be referred to later.

Selection of Method of Treatment.—Blind bouginage should be condemned as unsafe, uncertain and obsolete. The peroral string methods have given good results and are safer than pushing bouginage, but cannot possibly be as safe as the method of pulling up a tapered bougie of graduated sizes that we shall

describe later. Jackson's peroral esophagoscopic bouginage, where the flexible bougie in increasing sizes is passed by sight, is the safest and best method where gastrostomy is not done. The writer may be unduly optimistic, but, basing his conclusions on the hearty endorsement given this method by those who are using it, he feels confident that a fair trial of his retrograde bougie will convince any surgeon that it is unequaled in safety, rapidity of cure and permanency of its results.

**The Author's Retrograde Method.**—In the author's retrograde method gastrostomy, of course, is the first step, as in all retrograde methods of treatment. With the general improvement in the condition of the patient, due partially to the ample direct feeding, but especially to the freeing of the esophagus from the esophagitis and irritation secondary to stasis of food, the lumen usually opens sufficiently to allow the swallowing of a string. This achievement is the second essential in retrograde bouginage by the continuous string method.

In stringing the esophagus the method of string swallowing of Mixer, floating the string through while swallowing water, may be used. If this should fail, perforated shot or small steel balls, as recommended by von Acker, may be attached to the string to aid in passing it through. In the Bronchoscopic Clinic we have found the unknotted, slightly frayed end of the string best. In very small strictures a No. 15 buttonhole twist silk is used first. After it has been brought out through the abdominal fistula it is replaced by a heavier string, No. 4 Pearsal's twisted silk, which is the size usually worn. With small children a great deal of patience and perseverance are required on the part of both the physician and the nurse to get the string swallowed. With these little patients the string may be carried through the nose by means of a Bellocq cannula, as suggested by Socin and Mickulicz of von Acker's clinic, or a soft rubber catheter or No. 10 retrograde bougie may be used to carry it into the nasopharynx where it is worn. It is then introduced a few inches at a time through the anterior nares as the end in the pharynx is swallowed, thus preventing the child biting off the string. The child often regurgitates the string end, which is again put back and eventually swallowed. The proximal portion of the string, about a yard in length, is wound around a piece of adhesive plaster

used as a bobbin, and the intervening portion of the string is carried around the cheek and over the ear. The bobbin is fastened to the back of the neck, and the string and bobbin covered with adhesive to prevent the child pulling out the string. It may require several weeks' persistent effort to get the string through the stricture, especially if the lumen of the stricture is occluded by granulation tissue or is only intermittently open. After a length of string sufficient to allow the end to pass through the stomach has been swallowed, it is caught through the gastrostomy wound with a right angled, blunt, hook shaped Jackson pillar retractor (Fig. 2) and is thus brought out through the wound (Fig. 3). The other end is unwound from the bobbin and the ends tied together. If the string cannot be swallowed, a bougie may be passed up from below, by means of a retrograde esophagoscope, through the stricture into the mouth, and a loop of string attached and pulled down through the esophagus. This method has been very successful in a number of our cases. Chevalier Jackson, L. H. Clerf and the writer have been able to get a string through the obliterated part of an esophageal atresia by the two operators working at the same time, one performing the retrograde, the other the peroral esophagoscopy, getting the tubes close to the obliterated lumen both above and below. Fluoroscopic guidance being furnished by Dr. Willis F. Manges with his double plane fluoroscope, the retrograde esophagoscopist passed a bougie from below, and the tip was seized by the peroral esophagoscopist and drawn upward through the mouth. To this tip a string was attached and pulled down and through the abdominal fistula.

With the continuous string in place, the bougies are pulled upward from below, starting with the smallest size, No. 10. When attaching the bougie to the string a new string is also attached so that a fresh string may replace the old one at each sitting.

The bougie that I have devised is made of the best quality of rubber moulded on an endless braided silk thread (Fig. 1). A loop is left free at either end and the intervening portion is knotted to prevent its pulling out of the rubber. The ends are tapered so as to give a gradual dilating surface, and the sizes graduated from ten to thirty, French scale, 10, 12, 14, etc. The

length of the bougie is 35 cm., not including the loop at either end. This is sufficient to engage the full diameter of the bougie in the lumen of all the stricture levels simultaneously. Shorter and longer lengths and double tapered shapes have been tried, but the form here presented is the one that has proved most satisfactory (Fig. 1).

In adapting the mechanism of our methods of treatment the peculiarities of the esophagus as we see it in cicatricial stenosis have been considered.

1. As noted first by Billroth, the esophagus is dilated or bowl shaped above and funnel shaped below the stricture. This being the case, we can enter the stricture more easily from below, after passing the hiatus.

2. The strictures are frequently multiple, and at different levels, with eccentric lumina, as pointed out by Chevalier Jackson. With my bougie, all strictures, regardless of number, are dilated simultaneously.

3. Patients, especially children, are more tolerant of retrograde than peroral instrumentation and will allow the bougie to remain longer in position when pulled up from below than when brought down through the mouth.

4. The removal of the bougie upward, when the full size is engaged in the entire strictured area, requires less pull than downward removal, due probably to the reverse peristaltic action of the esophageal musculature excited by the movement of the bougie in the esophagus.

5. Retrograde bouginage by the continuous string method gives the maximum of safety, because the danger of rupture is avoided if proper care is used in the amount of force exerted in the pull on the string and if the bougie sizes are not "stepped up" too rapidly.

6. This method is carried out without anesthesia.

7. Hospitalization is not required for the carrying out of the bouginage.

This method is offered as an improvement, both as to the dilator and the technic used, on the Ochsner method of pulling upward a doubled rubber tube on the stretch, and at the Bronchoscopic Clinic we believe it to be the safest and most easily performed of all string methods of treatment.

Technic of Author's Method.—The continuous string is worn over the ear, and the child can attend school with it in

this position without inconvenience or annoyance from the other children (Fig. 4). In passing the bougie the patient is placed in a dorsally recumbent position. The feeding tube is removed and the string is cut in the middle of the loop from the nose to the gastrostomy wound. A new string is attached close up to the gastrostomy wound, and the lower end of the old string is joined to the loop of the bougie. A loop of string is now pulled through the mouth from the nasopharynx by using a Jackson pillar retractor, taking care not to pull the free nasal end back through the anterior nares. Upward traction is next made on the esophageal end of the bight by grasping it with one hand and passing the index finger of the other hand back over the base of the tongue. The string is then pulled taut over the tip of the index finger as a pulley, and the bougie is pulled upward in the axis of the esophagus into the lumen of the stricture. The force used in pulling the bougie into the stricture, although only a small amount be used, lessens the diameter of the bougie, and when the pull is relaxed the resiliency of the rubber gives the dilatation while the bougie is in place. It is left in position from fifteen to thirty minutes. After the patient becomes accustomed to the treatment, it gives very little discomfort, so that the little patients tolerate the presence of the bougie and often assist in pulling it through (Fig. 5). When the bougie is pulled through it is cut from the end of the string, the new string is pulled out through the nose as the old is removed, the ends are tied together and the new endless string is in place. The feeding tube is replaced in the gastrostomy wound and, although it is not used for feeding, it keeps the fistula from closing. The treatment is carried out once or twice a week, depending on the degree of reaction following, and the sizes are not "stepped up" until the bougie in use comes through very easily at a number of sittings. A bougie that comes through at one sitting without the use of undue force may meet with so much resistance at the time of the next treatment that a smaller size may have to be used. This is probably due to exacerbation of the chronic esophagitis which is present in many of these cases. After the step backward is made the forward progress is continued.

A fundamental point to be remembered in dilating cicatricial

strictures, as shown by Jackson, is that the scar tissue in many cases does not make up the entire circumference of the strictured lumen, and on dilatation the normal wall will stretch indefinitely if dilated gradually over a prolonged period, whereas, on sudden overstretching, the normal esophageal wall will rupture easily. When the periphery of a stricture is only partially cicatricial, Jackson states that it is the normal wall that dilates, not the cicatricial portion. The bearing of this in mind will make us doubly cautious in the amount of force used.

Case Reports.—The following cases of cicatricial stenosis of the esophagus have been treated in the Bronchoscopic Clinic of the Jefferson Hospital and the Bronchoscopic Clinic of the University of Pennsylvania Hospital by the method of continuous string, retrograde bouginage, using the author's bougie (Fig. 1).

In nineteen of these twenty-five cases the lesion was due to the swallowing of household preparations of caustic alkali by children whose parents, through carelessness or ignorance of the sinister nature of these dangerous preparations, had left the caustics within their reach; one case was the result of trauma due to unsuccessful attempts at removal of a foreign body before admission of the case to the Bronchoscopic Clinic; two cases were posttyphoidal in origin; the remaining three cases were adults whose burns were due to irritant poisons. One swallowed sulphuric acid by accident, another took bichlorid of mercury with suicidal intent, and the third swallowed household ammonia mixed with small pieces of glass in an attempt at suicide.

These cases are numbered and reported in chronologic order of their admission to the clinic. It will be noticed in a number of the earlier cases that treatment with the author's bougie was not begun until a considerable period of time had elapsed after admission of the patient to the clinic. This will be explained by the fact that the author's bougie was not devised until these cases had been under treatment for some time.

A tabulation of results attained under the author's continuous string retrograde bouginage shows that complete cure was obtained in three cases (12 per cent of the total number).

functional cure in nineteen cases still under observation and progressing rapidly to complete cure (76 per cent), improvement in three cases, two of which have been under treatment less than a month and the third of which died of intercurrent disease (12 per cent). Percentage of cases unimproved, zero.

Case 1.—Female, white, aged 23 months. Admitted to the Bronchoscopic Clinic three months after swallowing a preparation of household caustic alkali. The patient was able to swallow only small quantities of liquids, and these slowly. Dr. Chevalier Jackson performed an esophagoscopy, which showed a stenosis just below the cricoid level, with cicatrices on the posterior esophageal wall. Peroral esophagoscopy bouginage was done weekly. The patient was able to take sufficient liquid nourishment by mouth, but frequently developed attacks of dysphagia even for liquids. Following these, an attack of fever with bronchial symptoms occurred. Generally her nutrition was maintained and the child was very well nourished, but it was decided that progress was not satisfactory. On Roentgen ray examination Dr. Willis F. Manges reported as follows: "Almost complete occlusion of the esophagus at the level of the third dorsal vertebra with considerable proximal dilatation." Gastrostomy was done and esophagoscopy bouginage carried out for fourteen months, when a string was swallowed and retrograde treatment started with a No. 12 catheter. One month later an 18 catheter passed with slight difficulty. The sizes were increased slowly, the stricture being very resistant. In two weeks we were able to pass a No. 19 catheter with considerable difficulty. This was continued twice weekly for a month. At times it was necessary to use smaller sizes and the condition seemed stationary. The author's bougie was then used, starting with No. 18. In six weeks the bougie size had increased to 22. All soft foods were swallowed without difficulty. Ten weeks later we had progressed to easy passing of No. 26 bougie, when the child developed measles and the treatment was discontinued until recovery, when a No. 26 again was passed with slight resistance. Retrograde bouginage continued until, at the present time, a No. 28 bougie is passed without resistance, the child takes all food by mouth and is treated as an outpatient. The patient has a functional cure, but will be kept under observation and weekly treatment

for a considerable period of time because of the dense fibrous strictures.

NOTE.—All bougie sizes referred to in these cases are graded on the French scale.

Figure 8, print of Dr. Menges' Roentgen ray film, shows the condition of the stricture on admission of the patient.

Figure 9, Case 1, shows bougie No. 28 in position.

Case 2.—Female, white, aged five years, was admitted to the Bronchoscopic Clinic with a history of having swallowed lye seven months previously. The accident resulted in immediate dysphagia for solids and the child subsisted, losing much weight, on liquid diet. On admission the patient was suffering from influenza pneumonia and was regurgitating large amounts of the liquids she attempted to swallow. The child was transferred to the medical department of the hospital for treatment of the pneumonia and her general condition improved. Ten days later, after esophagoscopy, Dr. Chevalier Jackson reported, "Almost complete atresia. Lumen will not admit the smallest esophageal bougie." Gastrostomy was advised and Dr. DaCosta performed the operation, from which recovery was uneventful. Esophagoscopy was then done and showed a high level stricture in the esophagus. Esophagoscopic peroral bouginage was done weekly. The child improved rapidly, gained in weight and was able to take liquids by mouth. At the end of eight months the esophageal lumen has opened to such an extent that the child was taking soft food by mouth, and the gastrostomic feeding was discontinued, although the tube was kept in position to maintain the fistula. Eleven months after admission the patient was discharged from the hospital, to return as an outpatient for weekly bouginage. At times the child had trouble in swallowing and the mouth feeding would have to be supplemented by feeding through the gastrostomic tube. Her general condition was good, but the cure was not progressing as rapidly as was desired, so a string was swallowed and retrograde bouginage started with No. 10 catheter. This retrograde treatment was kept up at weekly intervals for six months, and the size had increased to No. 16, but progress seemed to be made no further. Two months later retrograde bouginage with the author's bougie, then newly devised, was begun and the sizes were



gradually increased from No. 16 until, eight weeks later, a No. 22 was being passed and the child was swallowing soft food easily. The child's general condition was good, and weekly bouginage under this method was continued. At the present time a No. 28 bougie is being passed without resistance, and the child is swallowing all kinds of food normally. This is a functional cure rapidly progressing to a permanent cure.

Case 3.—Male, aged  $2\frac{1}{2}$  years, drank lye seven weeks before he was admitted to the Bronchoscopic Clinic, swallowing very little liquid and in a decidedly emaciated condition. Dr. Jackson's esophagoscopy revealed an ulcerative esophagitis beginning at the pyriform sinus. The esophagoscope was not inserted through the ulcerated area. Roentgen ray examination showed a marked stenosis of the esophagus in the mid-dorsal region. In order to nourish the patient a gastrostomy was performed by Dr. Wm. Hearn, one week after admission. Weekly peroral esophagoscopy bouginage was used for one year, when the child's general condition was good, although at times aphagia developed, followed by bronchial symptoms with high temperature. This condition, due to aspiration of the overflow from the esophagus, would subside after the esophagus had opened up. When the dilatation had progressed to the use of a No. 20 filiform bougie, the continuous string bouginage with the author's bougie was instituted and carried out twice weekly. The child improved rapidly. No further attacks of dysphagia or bronchitis developed and, six months later, the little patient was readily passing a No. 26 retrograde bougie. He was then discharged from the hospital, to return for treatment, and at the present time, ten months later, he is still under observation and passes a No. 30 retrograde bougie without resistance. The child is cured functionally, is well in every way, and is attending school regularly. However, as a matter of precaution, the gastrostomy tube will be kept in place and a bougie passed occasionally until the scar tissue is entirely resorbed. This case presents one of the most difficult conditions to cure, a long tight strictured area in the middle of the esophagus.

Case 4.—Male, aged two years. Five months before his admission to the Bronchoscopic Clinic this child swallowed a

solution of lye. Immediate dysphagia for solids resulted, so that he afterward lived on liquid diet. The roentgen ray findings of Dr. Willis F. Manges showed a narrowing of the middle third of the esophagus with smooth lumen. The boy was treated as an outpatient with weekly esophagoscopy bouginage for seven months, when considerable difficulty in swallowing liquids developed and gastrostomy was done by Dr. Klopp. Subsequent esophagoscopy by Dr. Jackson disclosed severe esophagitis but no erosions. A small amount of blood came up through the lumen of the upper stricture. About 5 mm. beyond this a tighter stricture was felt with the esophagoscopic bougie. Esophagoscopy bouginage was again continued. Three months later, esophagoscopy showed the upper stricture well dilated and the treatment was continued. The mother was persuaded that she could care for the child with a string in situ, and a string was swallowed, recovered and made endless. Retrograde bouginage with the author's bougie was started, beginning with No. 18, which passed without resistance. Weekly passage of the bougie has been carried on. Now a No. 30 passes easily and the child is functionally cured.

Case 5.—Female, white, aged 8 years, admitted to the Bronchoscopic Clinic with history of having swallowed a jackstone two weeks before. Following this, three unsuccessful attempts at removal had been made, and the jackstone had been pursued downward in the esophagus to the level of the sixth dorsal vertebra. On admission, the child was swallowing only liquids and appeared quite ill, although apparently fairly well nourished. Dr. Thomas MacCrae made a physical examination and reported cardiac dilatation, advising rest as treatment. The child's general condition improved under rest and careful regulation of diet. Esophagoscopy examination by Dr. Jackson showed a jackstone tightly impacted in the tissue, apparently outside the esophageal wall. Gentle manipulation indicated that there was resistance downwards as well as upwards. There was an ulceration of the esophageal wall about 4 cm. above the present location of the jackstone. At this point a large flap of edematous mucosa covered the esophageal wall and projected out into the lumen of the esophagus, in form like a polypus, except for the raw surface which was downwards. The jackstone was surrounded by fungations and

shreds of lacerated tissue. The second esophagoscopy disclosed the strictured esophagus at the crossing of the left bronchus. A lobule projected into the lumen from the right. The foreign body was rigidly fixed, no lumen below it. Esophageal ulceration showed no tendency to heal since the previous inspection. The jackstone was removed by esophagoscopy by Dr. Jackson. The roentgen ray by Dr. Willis F. Manges showed practically complete obstruction of the esophagus. A week later the flap, visible at the first examination, appeared to have shrunk somewhat, but still had a loose extremity free in the esophageal lumen. Below that point conditions were very much the same as when first seen. No lumen was visible below the pocket in which the jack was found.

Following the removal of the foreign body the child still was able to take only liquids, and immediate gastrostomy was done by Dr. Thomas A. Shallow. Recovery following the gastrostomy was uneventful. Liquids were swallowed readily in two weeks. Retrograde esophagoscopic bouginage was carried out and some improvement was noted in swallowing. Efforts at string swallowing were unsuccessful and, two months later, a steel stemmed filiform bougie was passed retrograde, by means of the esophagoscope, through the stricture into the pharynx, a string attached, and was drawn into the stomach. Retrograde bouginage with catheter was now carried out regularly, and in six weeks No. 18 catheter was being passed and the child was swallowing soft foods, but sometimes there seemed to be contractions of the stricture between treatments and constant difficulty in swallowing. Retrograde treatment with the author's bougie was instituted and the lumen stayed open for longer periods. In five months the dilatation had reached No. 24. Six weeks later the sizes had been "stepped up" to No. 28, and the child was referred to the family physician, from whom reports note continuous improvement and ultimate cure.

Case 6.—Male, white, aged six years, admitted to the Bronchoscopic Clinic five weeks after swallowing a solution of lye. At that time the child was swallowing liquids with difficulty. His general condition was fair. Roentgen ray examination by Dr. Henry K. Pancoast showed constriction at the fourth cer-

vical vertebra and cardiac end of the esophagus. On esophagoscopy we found active bleeding ulceration just below the cricopharyngeus. Further exploration was deferred until after healing of the ulceration. Gastrostomy was performed by Dr. George P. Mueller to admit of ample feeding and to give esophageal rest. The patient's condition improved markedly after gastrostomy, and peroral esophagoscopy bouginage was carried out weekly. Two months later the patient was able to swallow liquids readily, and a string was swallowed and a No. 10 retrograde bougie passed. Bouginage was done twice weekly and the esophagus was dilated so that, six months later, a No. 20 slipped through easily. At this time gastrostomy feeding was discontinued, but the tube was still worn to maintain the gastrostomy opening.

The patient was discharged from the hospital to return as an outpatient for weekly bouginage. No. 22 passed easily, and the progress continued so that in six months we carried through a No. 28. Now a No. 30 passes without resistance, and the patient, in the presence of the doctor, pulls it through himself. This we consider a functional cure, the patient being well in every way, but he will be kept under observation and will have a bougie passed once or twice a month for at least a year.

Case 7.—Female, negro, aged two years. One year before her admission to the Bonchoscopic Clinic she drank a solution of lye. Following the accident, difficulty in swallowing developed, and the child was treated by blind bouginage by her family physician in a southern city. On admission she was greatly emaciated, her weight being  $13\frac{1}{2}$  pounds. She was swallowing only small quantities of liquid. Dr. George P. Mueller immediately performed a gastrostomy. In five weeks the child's weight had increased to  $17\frac{1}{2}$  pounds, due to the gastrostomy feeding, and her general condition was better. She was able to swallow liquids, and after about three months' persistent effort, with the aid of the nurse, we succeeded in getting her to swallow a string. Esophagoscopy bouginage had been carried out perorally up to this time. Roentgen ray examination by Dr. Henry K. Pancoast showed a long stricture in the cervical esophagus with a second strict-

ure at the level of the diaphragm. The smallest sized bougie met with resistance, but the stricture gradually yielded, bouginage being carried out twice weekly until the end of two months, when a No. 20 bougie passed readily and all feeding was done by mouth. The patient was discharged from the hospital and treated as an outpatient once a week by retrograde bouginage. At present a No. 24 is passed easily, and the child is functionally cured, although still under treatment.

Case 8.—Male, white, aged two years, admitted to the Bronchoscopic Clinic. Three months previous to admission the child drank a solution of lye. After the acute symptoms following the burn had subsided, the patient was able to take semisolids for about a week, when he developed aphagia. Gastrostomy had been done two months previous to admission by Dr. Oberlock, Pomfret, Conn. Attempts at treatment of the stenosis of the esophagus were made, but were unsuccessful, although the child gained in weight under the gastrostomy feeding, and on admission his nutrition was good. Immediate roentgen ray examination by Dr. Willis F. Manges disclosed complete stenosis of the esophagus about the level of the bifurcation of the trachea. Esophagoscopy by Dr. Jackson the next day showed scar tissue with patches of ulceration and granulation which completely obliterated the lumen of the esophagus at a point 14 cm. from the upper teeth. No lumen was found. Attempts were made at weekly intervals to find the esophageal lumen by peroral bouginage. Some dilatation of the upper stricture level was effected and a string swallowed two months following admission to the clinic. Retrograde bouginage started with No. 12, coming through with slight resistance; the sizes were gradually stepped up as the bougies came through easily until, at the end of two months, a No. 18 was passing, and the child was taking liquids very freely by mouth and his general condition was excellent. Shortly after this, however, the child developed an acute respiratory infection which progressed to bronchopneumonia, and the patient died after four days' illness with uremic symptoms complicating the pneumonia. It is probable that the ingestion of the caustic alkali, and especially the effect of the resultant water starvation on the kidneys, were causative factors in the death of the child. The retrograde bouginage was

progressing very satisfactorily, and we feel that it should be placed on the list of cases improved under this method of treatment.

Case 9.—Female, white, aged two years. Four and one-half months before her admission to the Bronchoscopic Clinic this child drank a small amount of caustic alkali solution. After the acute symptoms from the burn had subsided, she was able to take solid food at times but would have frequent regurgitation. For three months previous to admission the child was on liquid diet. Roentgen ray examination made at the Maine General Hospital showed a stricture of the esophagus. Gastrostomy was done and dilatation carried out, but the progress was not satisfactory and the child was referred to the clinic. On admission the patient's condition was good. Roentgen ray examination by Dr. Willis F. Manges showed a marked obstruction of the esophagus at the level of the articulation between the manubrium and the gladiolus. The diameter of the narrowest portion was about one-eighth inch and the proximal portion showed considerable dilatation. String swallowing was successful and retrograde bouginage was begun. In six weeks' time the sizes had increased to No. 20, and in three months more a No. 30 passed through with but slight resistance. This patient is functionally cured, but as a precautionary measure will be kept under occasional observation for a prolonged period.

Case 10.—Male, white, aged 27 years. Three months before admission to the Bronchoscopic Clinic this patient swallowed pieces of glass and several ounces of household ammonia with suicidal intent. Difficulty in swallowing began immediately and became progressively worse until nine weeks after this when gastrostomy was done. Roentgen ray examination by Dr. Willis F. Manges on admission showed marked and abrupt narrowing of the esophagus above the suprasternal notch. The barium column at its narrowest point was about one-eighth of an inch. This narrowed lumen extended for a distance of at least three and one-half inches. Esophagoscopy by Dr. Jackson revealed superficial ulceration just above the stricture located near the crossing of the left bronchus. The lumen of the stricture was about 3 mm. in diameter and somewhat encroached upon by granulations. A string was swallowed and

recovered and a No. 10 bougie pulled through with tight resistance. The bougie sizes were cautiously advanced and thirteen weeks later a No. 20 was passed easily. Feeding was done by mouth and the patient's weight increased to 152 pounds. At the end of a month a No. 28 was used with only slight resistance. The patient was discharged to return for treatment as an outpatient. Weekly treatments have been continued and the patient is functionally cured. When No. 30, the largest sized retrograde bougie, had passed several times without resistance the patient was taught to swallow the silk woven bougie, starting with No. 26. Now the patient readily swallows a No. 36 silk woven bougie, and the gastrostomy opening is maintained only for emergency. The man is cured but will be kept under observation for the possibility of cicatricial contraction. Parenthetically it may be stated here that, while we believe pushing down a bougie blindly is dangerous, an intelligent patient may be taught to swallow one with little or no risk in an esophagus free from ulceration or malignancy.

Case 11.—Female, white, aged four years. Three months before her admission to the Bronchoscopic Clinic the child drank a solution of lye. She was thereafter able to swallow only liquids. About one week before admission an attempt to dilate the stricture had been made by blind bouginage, but this had proved unsatisfactory and the child was referred to the clinic. At the time of admission, although the child was taking only liquids, her nutrition was fair. Roentgen ray examination showed narrowing of the esophagus from about one inch below the level of the suprasternal notch to within an inch of the diaphragm. Esophagoscopy diagnosis of ulcerative esophagitis and cicatricial stricture was made by Dr. Jackson. Gastrostomy was performed by Dr. Shallow and a string was swallowed. Weekly retrograde dilatation was instituted, and in ten weeks progress had been made to No. 16. Seven weeks later a No. 18 passed without difficulty. The patient was able to swallow all food by mouth, and her general condition was good. Eleven months after admission the patient was discharged, treatment to be continued by the family physician, a No. 26 retrograde bougie passing without difficulty. At the end of six months the patient returned for observation and a No. 30 was easily slipped through the stricture. A copy

of the Roentgen ray report of this date follows: "From the level of the upper border of the sternum to the stomach the esophagus has an almost normal lumen." This patient is now permanently cured and the gastrostomy wound will be closed.

Case 12.—Female, white, aged ten years, admitted to the Bronchoscopic Clinic twenty-two months after drinking a solution of lye. Following the accident she was able to take only liquids, and for nearly a year was treated three times a week by blind bouginage without any improvement. She was then taken to a second physician, who treated her for nearly a year by the same method. At times she would be able to swallow soft food and at other times would have great difficulty in swallowing even liquids. Complete aphagia developed and gastrostomy was performed. The inability to swallow liquids persisted for several weeks after the gastrostomy. Esophagoscopy was attempted under ether, and it was stated that an upper stricture was dilated but that there was a lower one, tightly closed, upon which no attempt was made at dilatation. On admission the patient seemed well nourished but underdeveloped. Dr. Manges' report of Roentgen ray examination, one week after admission, reads: "There is practically complete obstruction of the esophagus at the level of the suprasternal notch. The stricture is distinctly at the anterior portion of the esophagus." On esophagoscopy Dr. Jackson found that the folds crowded in at the bottom of the dilated portion of the thoracic esophagus, near the junction with the cervical esophagus. No opening was found at this examination. Weekly esophagoscopies were performed, and in ten weeks the patient was able to swallow liquids without discomfort. A string was swallowed and retrograde dilatation was begun with No. 10 bougie passing with considerable difficulty. Five weeks later a No. 14 passed easily, and the sizes advanced about once in two weeks until a No. 20 passed with very slight resistance. At the present time, 18 months after the beginning of treatment, the patient is getting all her nourishment by mouth. She has been referred back to the family physician, who is carrying out the retrograde treatment. A recent report from the family physician states that progress has been favorable. As the patient is swallowing all kinds of food she may be said to have a functional cure.



Case 13.—Male, white, aged fourteen years. Two months before admission to the Bronchoscopic Clinic the boy developed typhoid fever and was in bed four weeks. Dysphagia for solids developed during convalescence, and on admission he was swallowing only liquids, and those with difficulty. He was emaciated, pallid and feeble and had an anxious expression on his face. He took water into his mouth and rejected it instantly; he held his hands in running water to relieve his thirst. On account of this severe water starvation immediate gastrostomy was done under local anesthesia by Dr. Thomas A. Shallow. Uneventful convalescence followed the gastrostomy and he took liquids freely by mouth, although occasional dysphagia occurred. Roentgen ray examination showed complete obstruction below the suprasternal notch. The proximal portion of the esophagus was somewhat dilated. The lower border of the barium column was smooth. Esophagoscopy by Dr. Jackson disclosed the scar in the esophagus beginning at the level of the lower border of the cricoid cartilage, broadening and extending downward, ending in a cicatricial stenosis about 4 cm. below the cricopharyngeal fold. There was no evidence of foreign body. Diagnosis of post-typhoidal cicatricial stenosis of the esophagus was made. A string was passed through the stricture ten weeks after admission. No. 10 retrograde bougie met with firm resistance, when traction was made, passed only partially through and was withdrawn. Biweekly seances of bouginage were carried out, and in three weeks a No. 10 retrograde bougie passed with ease. This semiweekly bouginage has been continued up to the present time. After the first two months' treatment the patient was able to swallow all well masticated food and is now accepting a No. 28 bougie with very slight resistance. The dilatation has been very slow. There are three distinct stricture levels and, while the patient has a functional cure, he will be kept under observation for some time, the gastrostomy opening being maintained for emergency on account of the large amount of cicatricial tissue in the esophagus. The boy is perfectly well in every way and has been attending school regularly during the last ten months. He wears his endless string without annoyance.

Case 14.—Female, white, aged two years, put a small quan-

tity of lye into her mouth six weeks before her admission to the Bronchoscopic Clinic, and as a result of the lye burns only liquids could be swallowed. However, this patient's general condition was fair. Esophagoscopy by Dr. Jackson revealed the ulceration just below the crossing of the left bronchus, below which the esophagus was swollen shut. No bougies were passed and gastrostomy was advised because of the extent of ulceration and was done by Dr. Thomas A. Shallow. Considerable difficulty was experienced in getting the child to tolerate a string for swallowing. The string finally passed after a month of effort and was recovered and the author's method of retrograde bouginage started with a No. 10 bougie. In five weeks a No. 20 traversed the esophagus and six months later a No. 28 passed without resistance, and the patient was sent home functionally cured, with instructions to the family physician to maintain the gastrostomy opening and to keep the condition under observation by the occasional passing of a bougie.

Case 15.—Male, three years of age, admitted to the Bronchoscopic Clinic three weeks following the ingestion of lye. Dysphagia for solids resulted immediately, and on admission to the clinic the child was taking only small quantities of liquid. He was pale and markedly decreased in weight but otherwise in fair condition. However, because of his need for nutrition, a gastrostomy was done by Dr. Thomas A. Shallow, and after an uneventful recovery the child's condition improved greatly. Roentgen ray examination showed a stricture in the middle and lower thirds of the esophagus. Six weeks after admission a string was swallowed and No. 10 retrograde bougie passed. In three weeks the sizes had increased to No. 18, and the patient, whose condition was excellent, was allowed to leave the hospital and return for semiweekly bouginage. At the end of a month dilatation had progressed to No. 22, and the patient was sent to his home near Pittsburgh, with instructions to report to Dr. Ellen J. Patterson for continuation of treatment. Dr. Patterson reports that retrograde bouginage has been kept up and at the present time the child is well, with normal lumen, but is kept under observation and occasional treatment.

Case 16.—Female, white, aged two years. The child was admitted to the Bronchoscopic Clinic twelve weeks after she had swallowed a solution of lye. Home remedies were instituted, but a physician was not consulted until a week after the accident, when the parents noticed that the child was regurgitating her food. Roentgen ray examination about six weeks after the accident by Dr. R. W. Holmes of Chillicothe, Ohio, showed a stricture of the esophagus. The child was then referred to Dr. J. F. Baldwin who performed a gastrotomy. Following this operation, which rendered possible the giving of regular and sufficient nourishment, the child improved rapidly, and the esophageal rest ameliorated the condition of the esophagus so that the patient was able to take liquids slowly by mouth. The child was referred to the clinic by Dr. Baldwin for treatment of the stricture. On admission the patient's general condition was fair and she could swallow liquids slowly. The abdominal wound showed the gastrotomy wound healing nicely with the tube in situ. String swallowing was successful at the end of a week and retrograde bouginage was started, the esophagus permitting to pass readily a No. 14 bougie. Sizes were cautiously stepped up until a No. 26 retrograde bougie was being passed. The child was referred back to her home, near Columbus, the retrograde treatment being continued under the direction of Dr. Baldwin. After No. 30 had passed successfully the treatments were done at longer intervals. A letter recently received from Dr. Baldwin reports that the child is cured and the gastrotomy wound has been closed.

Case 17.—Female, white, aged 22 months. Admitted to the Bronchoscopic Clinic nine weeks after she had drunk a solution of household lye. When the acute symptoms of the burn had subsided it was discovered that she could swallow only soft foods, and this dysphagia increased until, at the time of admission, she took only liquids, sometimes freely, sometimes with difficulty. Gastrotomy was done by Dr. Shallow. Roentgen ray was taken, but the extent of the stenosis was not discovered. There was no evidence of foreign body. Dr. Jackson performed an esophagoscopy, which showed a tight stricture with cicatricial margins, about 4 cm. below the cricopharyngeus. No granulations or ulcerations were visible. Five

weeks after gastrostomy the patient was able to swallow liquids with difficulty, but dysphagia for solids still existed. Two weeks after this, string swallowing was successful and a No. 10 bougie pulled through. In three weeks more a No. 12 passed. At the end of two months the bougie sizes had increased to No. 22. Ten weeks later a 26 passed easily. The child was found to have a positive diphtheria culture and was transferred to the Municipal Hospital. When the child had fully recovered and was readmitted to the Bronchoscopic Clinic the retrograde treatment was continued until a No. 30 passed. The child is in condition to be referred home to be kept under observation by her family physician. She has a functional cure which has every indication of being a permanent one, but the time is too short to pronounce her a permanent cure requiring no further observation. Fig. 12 shows No. 30 bougie in situ.

Case 18.—Male, white, aged three years. Six months before admission to the Bronchoscopic Clinic the child had drunk a solution of lye. Dysphagia for solids came on about a month after the accident and progressed until, three months after the accident, the patient had great difficulty in swallowing and for a time could not swallow even liquids. This, however, improved and liquids were taken readily. The following two months the patient was under the care of a physician who used blind bouginage at weekly intervals, but no improvement was noted and the child was referred to us. On admission the Roentgen ray examination by Dr. Willis F. Manges showed a tight stricture in the upper third of the esophagus. Esophagoscopy showed that about 3 cm. below the cricopharyngeus there was an ulcerated area with granular borders and a very large area of esophagitis surrounding the lesion. Below the ulcer the esophagus was narrowed to a lumen of about 1 mm. by cicatricial tissue. The extensive ulceration and esophagitis necessitated immediate and complete esophageal rest, and Dr. Shallow performed a gastrostomy. One month later a string was swallowed and a No. 10 bougie pulled through with slight resistance. At the end of five weeks the size used was No. 18, and the child took soft food readily by mouth. In another month dilatation had

reached No. 24, and the patient was allowed to go home, the family physician continuing the treatment.

Case 19.—Male, white, aged 41 years, accidentally took a mouthful of sulphuric acid six months before admission to the Bronchoscopic Clinic. Diagnosis of cicatricial stenosis had been made from the history and Roentgen ray studies and gastrostomy, followed by unsuccessful attempts at retrograde dilatation, done before admission to Jefferson Hospital. The man's weight had been maintained by the gastrostomy feeding. Esophagoscopy by Dr. Jackson revealed dilatation of the cervical esophagus with stricture, the lumen about 3 mm. in diameter at bottom, with superficial erosions but no deep ulcerations and no diverticulum. Roentgen ray examination by Dr. Willis F. Manges showed a lumen one-eighth inch back of the manubrium. The second stricture was at the level of the seventh vertebra. A string was swallowed and recovered through the gastrostomy opening, and retrograde treatment with the author's bougie started. Increasing sizes of bougies were used twice weekly, and in three weeks a No. 26 bougie passed easily and the patient took well masticated food by mouth and was sent home, the treatments to be continued by the family physician, using the same method.

Case 20.—Male, white, aged 2 years, admitted to the Bronchoscopic Clinic three weeks after drinking a solution of lye. The child had not been able to swallow solid or soft food since the accident and regurgitated all but small quantities of liquid. Roentgen ray examination showed a narrowing of the esophagus at the juncture of the middle and lower thirds; solution of bismuth passed through, in a very narrow stream. The child was unable to get sufficient nourishment and gastrostomy was done immediately. Three days later the patient swallowed liquids more easily, but one month later esophagoscopy showed the esophagus still inflammatory and ulcerated, with sloughing in the thoracic esophagus. When swallowing had improved, a string was passed through the stricture and recovered through the stomach. Retrograde dilatation was started, No. 10 bougie coming through easily. Increased sizes were used up to No. 16, which met with resistance. Retrograde treatment was continued at two sittings a week. Two months from the beginning of retrograde bouginage the esophagus

is permitting to pass a No. 24, and the patient is taking all food by mouth. This case is progressing very rapidly, and we shall be exceedingly careful not to overdilate the esophagus, as the duration of the condition has not been long enough for dense cicatricial tissue to form, and we should not expect to get much resistance to the passage of the bougie. The case can be classed as a functional cure and will be kept under close observation for at least a year, with weekly or biweekly bouginage and, later, esophagoscopy inspection of the strictured area before the case is discharged. The gastrostomy opening will be maintained until the child is entirely recovered.

Case 21.—Male, white, aged 20 years, farmer by occupation, was admitted to the Bronchoscopic Clinic with the history of having developed typhoid fever eighteen months previously, with which he had been ill five weeks. During convalescence and afterwards he had difficulty in swallowing solid food. This dysphagia became progressively worse until he was able to take only liquids. During the following year and a half he subsisted on liquids almost entirely. Occasionally there would be a time when he could take soft food, and after these attempts he would be unable to swallow even liquids. At the time of admission he weighed 115 pounds. Before his illness his best weight had been 162 pounds. He had not had treatment for the esophageal condition. Except for his underweight his condition was fair. Roentgen ray examination showed a marked obstruction of the lower end of the esophagus, the narrowest lumen being about one-fourth of an inch in diameter. The outline of the barium conformed to the cicatricial type. Gastrostomy was done by Dr. T. A. Shallow, under local anesthesia. Two weeks later esophagoscopy showed that the cervical esophagus was small in diameter for the age of the patient. The thoracic esophagus was normal until the level of the heart was passed. About 3 cm. above the diaphragm a cicatricial stenosis was encountered with a lumen about 3 mm. in diameter. Diagnosis was posttyphoidal cicatricial stenosis of the esophagus. A string was swallowed immediately and retrograde bouginage started with No. 10 bougie, to which very slight resistance was offered. The sizes were increased until a No. 16 met with a slight pull. Semiweekly bouginage has been kept up until the present time, two months after the

beginning of retrograde bouginage with the author's bougie, when a No. 30 passes easily. This patient will be kept under observation for at least a year and will be taught to swallow olivary fiber bougies, starting with No. 26 and continuing on above 30, the largest retrograde bougie. The gastrostomy opening will be maintained. This being a cicatricial stenosis of long standing, the case will not be discharged from observation until we are sure that a permanent cure has been obtained.

Case 22.—Male, negro, aged three years. One month before admission to the Bronchoscopic Clinic the patient drank a solution of lye. He had been able to swallow only liquids from that time. The night before admission the child attempted to swallow a piece of meat and complete aphagia resulted. On admission the child was poorly nourished, quite ill, skin warm and dry, tongue heavily coated, breath foul. Esophagoscopy by Dr. Clerf showed constriction of the esophageal lumen at the suprasternal notch. There was some ulceration. A No. 10 steel stemmed filiform bougie was passed through the esophagoscope into the uppermost stricture, very gently, and met with resistance.

Gastrostomy was performed at once by Dr. Righter under local anesthesia. Three weeks after admission the child swallowed a string, which was recovered through the gastrostomy opening, and retrograde bouginage started, No. 10 bougie coming through with slight resistance. In ten days a No. 14 passed easily, and one week later a No. 16. The sizes had advanced to No. 22 one month from the beginning of treatment, and from then on the patient took all food by mouth. He was allowed to go home under arrangement to return for semiweekly treatment. Now, six weeks after admission, a 26 has been passed. The gastrostomy tube will be kept in place and this method of bouginage continued weekly. The child has a functional cure but will be kept under close observation for at least a year and a half.

Case 23.—Female, white, aged 25 years, admitted to the Bronchoscopic Clinic with a history of scarlet fever and typhoid fever in childhood, tonsillectomy at 13 years of age, appendectomy at 17, cholecystectomy at 19, hospitalized with a diagnosis of hysteria with gastrointestinal symptoms at 20.

At 21 years, gastroenterostomy was done, and six months later a laparotomy for constrictions of gastroenterostomy opening. At 22 years of age she was again hospitalized for abdominal symptoms. At 24 a second tonsillectomy was done.

Because of the persistent gastrointestinal symptoms of which she was not relieved, she was in a melancholic state, and as a consequence took seven and a half grains of bichlorid of mercury. An acute nephritis developed, but from this she recovered. A week later she began to regurgitate food. Because of this a gastrostomy was done. A diagnosis of stricture of the esophagus was made and dilatation by a peroral method with a swallowed string was carried out over a period of six months at a hospital. On admission to the Bronchoscopic Clinic the patient was unable to swallow her saliva, and feeding was done entirely through the gastrostomy tube. An attempt was made to get her to swallow a string at intervals, as there were times when the saliva seemed to pass through the esophagus. These attempts were unsuccessful, and a retrograde esophagoscopy was done and a No. 8 steel stemmed filiform bougie was passed into the lumen of the stricture from below by sight. The patient was placed in the low stage of Jackson's high-low esophagoscopic position. The bougie was carried straight into the pharynx without resistance, the tip being brought out through the mouth. A small silk thread was attached and was pulled through the stricture into the stomach as the bougie was withdrawn. A No. 10 retrograde bougie was pulled through from below without resistance, and twice weekly the retrograde dilatation was repeated, the bougie sizes being increased with care until at the present time, six weeks after the beginning of treatment with the author's bougie, a No. 24 passes easily. The patient is able to take all nourishment by mouth, and as far as the esophagus is concerned has a functional cure. Her gastrointestinal symptoms persist and she is under the care of an enterologist. The progress of the treatment of her cicatricial stenosis has been very rapid and satisfactory, and from present indications will result in a complete cure within a few months.

Case 24.—Male, white, aged three years, admitted to the Bronchoscopic Clinic soon after the drinking of a solution of Iye. Dysphagia began shortly after the swallowing of the caus-



tic and increased until at the time of admission to the hospital the child took liquids with great difficulty. He was pale and anemic, ribs and clavicles prominent, would lie in dorsal decubitus, completely relaxed, saliva drooling from his mouth, in an advanced stage of water starvation. Because of this excessive dehydration, immediate gastrostomy was done. The child improved rapidly when sufficient nourishment was supplied by gastrostomy feeding. String swallowing was attempted at intervals when the child appeared to be swallowing saliva, but the effort was unsuccessful, the child refusing to cooperate. Retrograde esophagoscopy was done, using a 5 mm. Jackson retrograde esophagoscope. The lower limits of the stricture in the midthoracic esophagus were found and a No. 8 filiform steel stemmed bougie introduced into the lower lumen of the stricture. The child was placed in the low stage of the Jackson position for esophagoscopy, and the bougie gently insinuated into the stricture, and passed upwards into the pharynx, the filiform tip being brought out into the mouth from the pharynx with a Jackson pillar retractor through the stricture and through the gastrostomic fistula. The author's retrograde bougie, No. 10, was pulled through with slight resistance. The sizes have been stepped up until now a No. 20 is being passed and the child can take water by mouth. A start has been made in the treatment of this case, and we expect to continue it for at least a year, but a functionally normal esophagus, which means much to the child and to everyone concerned in its care, we hope to attain within a period of three months.

Case 25.—Female, white, aged three years, was admitted to the Bronchoscopic Clinic three months after drinking a solution of lye. Difficulty in swallowing was not noted until about a week after the accident. A physician was consulted at this time, and he treated the child with bismuth and olive oil. A grain of corn lodged in the esophagus, and liquids would not pass until several days later, when she regurgitated the grain of corn. Dr. Hair discovered a cicatricial stricture upon Roentgen ray examination and referred the child to the Bronchoscopic Clinic.

On admission the child did not appear ill, seemed fairly well developed and well nourished and swallowed liquids slowly

and with difficulty. Fluoroscopic and Roentgen ray studies by Dr. Pancoast confirmed the report of stricture of the esophagus in the middle third, from the lower border of the seventh to the second thoracic vertebra. Esophagoscopy showed bleeding, ulceration and granulation, with some scar tissue formation and a marked stricture of the esophagus at the level of the second dorsal vertebra.

Dr. Mueller made a gastrostomic fistula, and ten days later a string was swallowed, No. 10, 12 and 14 retrograde bougies being pulled through, No. 14 meeting with slight resistance. Bouginage was performed twice weekly until a No. 18 passed easily, and the child is now taking liquids and soft food by mouth. The treatment in this case, although progressing favorably, is just beginning and will be carried out with the utmost confidence in our ability to obtain a permanent cure.

#### CONCLUSIONS.

1. The great truth pointed out by Trousseau, that sooner or later all cases of esophageal stricture died of the bougie is a clear indication of the dangers attending the blind pushing downward of peroral bougies.

2. Esophagoscopy bouginage of cicatricial stenosis is relatively safe in careful hands, but its results are obtained slowly, especially in cases of multiple, eccentric strictures which have to be dealt with carefully and one at a time.

3. Rapid instrumental dilatation of cicatricial esophageal stenosis is dangerous.

4. It requires no argument to demonstrate that drawing a bougie through a strictured esophagus by a string which the bougie must follow is incomparably safer than pushing any bougie could possibly be. The only possible danger would arise from the mistake of violently pulling through an oversized bougie.

5. It is true that retrograde treatment requires a gastrotomy, but this is a relatively minor operation that can be done quickly and, if necessary, under local anesthesia. Moreover, the giving of all food by mouth being stopped, the esophagus is relieved from the static esophagitis which is so often a factor in increasing the amount of cicatricial tissue, the subse-

quent contraction of which renders the stricture more and more difficult to cure.

6. Though esophagoscopy is always advisable for determination of the condition of the esophagus before beginning treatment, once the endless string is in place, neither retrograde nor peroral esophagoscopy is necessary. It can therefore be carried on to complete cure by any careful practitioner.

7. With the bougie shown here, treatment of cicatricial stenosis of the esophagus is, I think, safer than by any other method, and the curative results are obtained more quickly than by any other justifiable method.

8. Hospitalization is not usually required except for gastrostomy in cases in which this operation has not already been done. The string is worn through the nose without annoyance, and children can attend school if it is so desired.

9. My coworkers have tested the method, and all have assured me that it is the greatest step forward yet made in the treatment of cicatricial stenosis of the esophagus. It is with the hope that others may find it of equal service in curing these little sufferers that my method is offered.

#### BIBLIOGRAPHY.

1. Carmody, T. E.: "Stenosis of the Esophagus," *New York Medical Journal*, 1921, CXIII, p. 427.
2. DaCosta, J. C.: "Modern Surgery," 7th Edition, pp. 930-937.
3. Eiselsberg, Vienna: "Endless String Sounding in Strictures of the Esophagus," *Deutsche med. Wochenschrift*, 1898.
4. Fletcher, R.: "On Strictures of the Esophagus and the Danger of the Bougie Therein," *Medico-Chirurgical Notes*, 1831, pp. 26-42.
5. Graham, D. W.: "Esophageal Strictures," *Canadian Medical Journal*, Feb., 1921, p. 120.
6. Guisez, J.: "Pathology and Forms of Inflammatory Stenosis of the Esophagus," 1922. (Extrait de la Gazette des Hôpitaux, March 7, 1922.)
7. Guisez, J.: "Diagnosis and Treatment of Strictures of the Esophagus and of the Trachea," 1923, p. 1-112.
8. Hacker: "Endless String Sounding for Cicatricial Stenosis of the Esophagus," *Wiener klinische Wochenschrift*, Nos. 24 and 26, 1894.
9. Henle, A.: "Treatment of Esophageal Strictures," *Centralblatt für Chirurgie*, Dec., 1922, p. 1850.
10. Jackson, Chevalier: "Peroral Endoscopy and Laryngeal Surgery," *Textbook*, 1914, pp. 522-539.
11. Jackson, Chevalier: "Cicatricial Stenosis Following the Swallowing of Caustic Alkalis," *Journal A. M. A.*, 1921, Vol. 77, No. 1, pp. 22-23.

12. Jackson, Chevalier: "Bronchoscopy and Esophagoscopy," Textbook, W. B. Saunders Co., 1922, Chap. 32, pp. 251-260.

13. Jacobson, Carl: "Importance of Deglutition and the Peristaltic Movements of the Esophagus in the Treatment of Stricture of the Esophagus," *Acta Oto. laryngol.* 3:146, Upsala, 1921.

14. Keene, W. W.: "An American Textbook of Surgery," 1904, pp. 759-766.

15. Keene, W. W.: "Surgery," Vol. 3, pp. 807-823.

16. Kurtzahn, H.: "Dilatation of Esophageal Stenosis," *Zentralblatt für Chirurgie*, Leipzig, Mar. 10, 1923, p. 381.

17. Ledoux, L.: "Stricture of the Esophagus from Burns," *Oto-Rhino-Laryngol. Internat.*, Lyon, 6:32, June, 1922.

18. Lotheissen, G.: "Esophagoplastics," *Beiträge zur klinischen Chirurgie*, Tübingen, 1922, Vol. 126, p. 490.

19. Mosher, Harris, P.: "Cardiospasm," *Penna. Med. Jour.*, 26: 240-249, January, 1923.

20. Mosher, Harris P.: "The Diseases of the Esophagus," in Loeb, "Operative Surgery of the Nose, Throat and Ear," Vol. 1, pp. 231-242.

21. Ochsner and Percy: "New Manual of Surgery," 1917, pp. 420-428.

22. Seiffert, A.: "Bougination in Severe Stenosis of the Esophagus," *Monatschr. f. Ohrenhkl. u. Laryngo. Rhinol.*, 55:1634, Berlin, Nov. 1921.

23. Sippy, B. W.: "Diseases of the Esophagus," in *Therapeutics of Internal Diseases*, Forcheimer, 1913, Vol. 3, pp. 47-66.

24. Stupka, Walther.: "Diphtheria of the Esophagus and Its Sequels," *Deutsch. Zeit. Chir.*, 170:1, Leipsic, April, 1922.

25. Zimanyi, V.: "Prophylaxis of Lye Accidents," *Münchener medizinische Wochenschrift*, Munich, June, 1923, Vol. 170:772.



FIGURE 1.

Bougie devised by the author for the retrograde treatment of esophageal stenosis. The rubber is vulcanized onto a braided silk loop with spaced knots. Various shapes were tried, and this one was found to answer all requirements in the most satisfactory way.



FIGURE 2.—JACKSON PILLAR RETRACTOR.

Used for recovering the swallowed string through the gastrostomic fistula and for withdrawing a loop of string through the mouth, from the pharynx, preliminary to pulling up the bougie, in the author's retrograde method of bouginage.



FIGURE 3.

The swallowed string is recovered through the gastrostomic fistula by the use of a Jackson pillar retractor. A loop is withdrawn, and, if the end has passed into the intestines, that side of the loop is cut, and the cut end is joined to the string on the bobbin, making the string continuous.



FIGURE 4.

Photograph of boy showing the method of wearing the continuous string over the ear. The gastrostomic tube, worn in situ, has been removed preparatory to treatment.



FIGURE 5.

Photograph of patient pulling the bougie into the stricture. The loop of string has been cut. A new string has been attached to the old one close to the gastrostomic fistula; the bougie has been attached to the end of the old string. A loop of string has been withdrawn through the mouth from the pharynx, and the patient is making traction on the abdominal end of the loop.



FIGURE 6.

Group of children with cicatricial stenosis of the esophagus due to caustic alkali burns. All were treated by the author's retrograde bougie. Number 1 is reported as Case 6, number 4 as Case 25, and number 5 as Case 7.





FIGURE 7.

Another group of children with cicatricial stenosis of the esophagus due to caustic alkali burns, all of whom were treated by the author's retrograde bougie. Number 2 is reported as Case 22, number 3 as Case 24, and number 4 as Case 20.

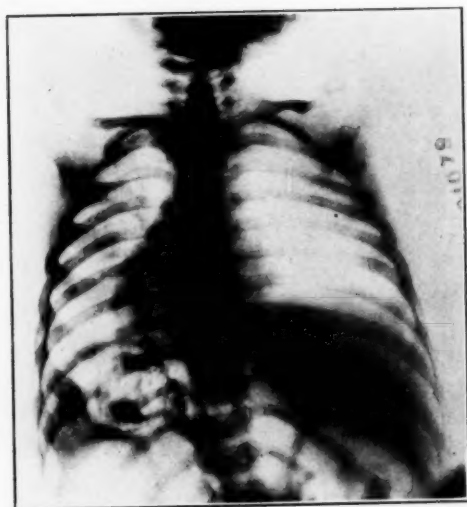


FIGURE 8.

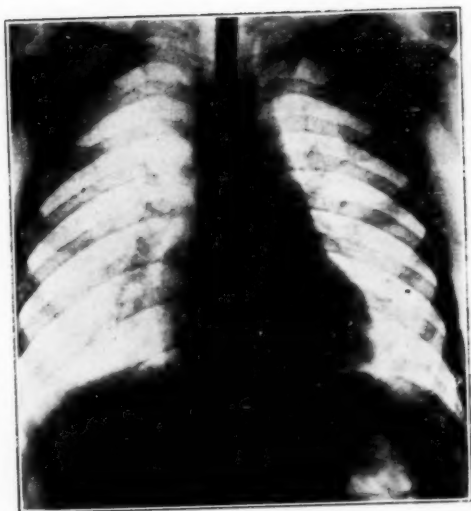


FIGURE 9.



CASE 7, FIG. 10.  
 Photograph of child on admission, following gastrostomy.



CASE 7, FIG. 11.  
 Photograph of same child eight months after the beginning of retrograde treatment. Functional cure has been obtained and child is treated as an outpatient.

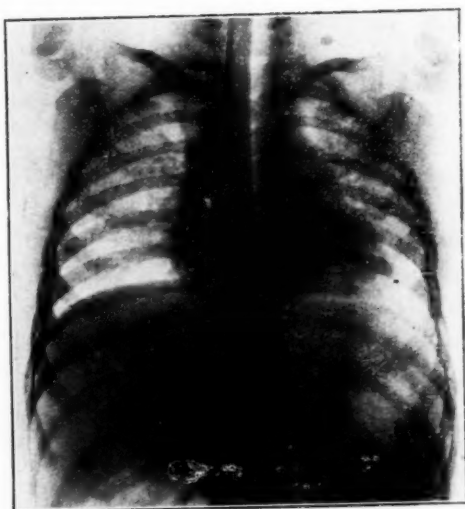


FIGURE 12.

FURTHER OBSERVATIONS ON THE HISTOPATHO-  
LOGIC CHANGES IN EXCISED FAUCIAL  
TONSILS.

By HOWARD H. BELL, M. D.

DEPARTMENT OF PATHOLOGY, WASHINGTON UNIVERSITY SCHOOL  
OF MEDICINE.

SAINT LOUIS.

In a preceding number of this journal I described briefly some of the lesions occurring in faucial tonsils. I used the classification of Semon and Williams,<sup>1</sup> which is as follows:

I. Acute Lacunar Tonsillitis—refers to inflammation confined to the lining of the crypts, and characterized usually by focal microscopic ulcerations with accumulation of chiefly polynuclear cells and fibrin formation.

II. Acute Parenchymatous or Follicular Tonsillitis—refers to inflammation of the centers of the follicles.

III. Acute Peritonsillitis or Quinsy—refers to inflammation of the peritonsillar mucous glands.

IV. Chronic Lacunar Tonsillitis—refers in general to widely dilated crypts containing masses of bacteria, showing irregular epithelium and invasion of lymphocytes.

V. Fibrosis of Tonsil.

VI. Chronic Peritonsillitis.

It is my present purpose to emphasize certain details not given in the preceding paper.

In the majority of tonsils removed routinely, one observes deep hematoxylin staining material which is without definition, and located very often beneath a break in the epithelium, or about follicles, or elsewhere in the tonsils. This material has been described in certain instances as fibrin. I would like to emphasize that it is as a rule not intimately associated with acute polynuclear reactions where one would expect much fibrin to occur. On the contrary, it is associated with lymphocytes.

It occurs very often where the epithelium is thin (See Fig. I) and appears as a dark heavily stained zone of ill defined material. Polynuclear cells are conspicuously absent. It is no doubt, as you will see, a very early lesion of acute inflammation, or the result of a stimulus insufficient to excite a more violent inflammatory reaction.

Necrosis of the type common in lymphoid tissue in general occurs also in the tonsils. (See Fig. II).

This deep hematoxylin staining material often appears surrounding necrotic areas in the centers of the follicles and is frequently crescentic in arrangement, and is frequently located on the side of the follicle toward the lacuna (See Fig. III). Regeneration develops from the centers of the lobules. Regeneration may be complete, which appears to be the rule, unless new capillaries are formed.

The deep hematoxylin staining material is often radially arranged (See Fig. IV). In order to study this deep hematoxylin staining material, very thin sections are required. On account of calcium deposit, which is common in tonsils, it is difficult to prepare suitable sections. Suitable sections, however, will show this deep hematoxylin staining material to be composed of distorted and fragmented nuclear material; pyknosis and karyoklasis are marked and the accumulated nuclear material takes a deep stain. (See Figs. V and VI).

Many of the cells are markedly elongated and spindle shaped. In more suitable preparation (See Fig. VII) cells resembling lymphocytes undergoing spindle-like elongation can be observed. This deep hematoxylin staining material appears to result from degeneration especially of lymphocytes which through pyknosis and karyoklasis permit this accumulated nuclear material to show in thick sections as ill defined deep staining material.

Fibrosis of Tonsil.—Not infrequently a fibrosis which appears to be progressive in character is observed in tonsils (see Fig. VIII). Such lesions have at times been considered tuberculous. Foreign body giant cells are occasionally present. Necrosis in lesions referred to is absent. I have not made the diagnosis of tuberculosis in such tonsils.

Diagnosis of primary tuberculosis in tonsils has usually been based on histopathologic study. Weller<sup>2</sup> by such means found

2.35 per cent of tonsils to show tuberculosis. He found caseation conspicuously infrequent, although other characteristics of the tubercle were present in his sections. Mitchel<sup>22</sup> studied the type of bacillus causing primary tonsillar tuberculosis in children and found the bovine type in 20 instances and the human type in six instances. In the experiences of Park and Krumwiede<sup>4</sup> and Griffith<sup>5</sup> the incidence of bovine type in tuberculous cervical adenitis is relatively great in children and decreases as age advances, which corresponds with Mitchel's observations in tonsils.

Calcium deposit in tonsils adds greatly to the difficulty of preparing sections. One will not infrequently find calcium deposit in the masses of bacteria and debris in crypts (see Fig. 9.) Direct examination of this material will frequently reveal partial calcification.

Clinical Acute Lacunar Tonsillitis.—I refer to the condition characterized by plugs of fibrinopurulent exudate protruding from the crypts associated with marked constitutional disturbances. In such tonsils the entire crypts are filled with exudate (see Fig. X). The parenchyma contains many polynuclear and numerous large mononuclear cells. The lymphocytes appear to be decreased in number. While this represents acute lacunar tonsillitis in type of reaction, it is, nevertheless, in extent distinctive from the focal lesions observed in tonsils removed routinely.

Striated Muscle in Tonsils.—It has been repeatedly pointed out that striated muscle frequently occurs in the capsule of tonsils, and occasionally extends along the trabeculae for a short distance. Striated muscle has been found in a few instances in the lymph follicles (see Fig. XI), though this association is very rare. Maclachlan<sup>6</sup> refers to the possible effect of muscle in the tonsil on emptying the crypts through compression. Such action, if present, is very ineffective, as shown by gross dissection of tonsils.

Tonsils of Infants at Term.—The tonsils of infants at term do not, in my experience, protrude into the pharynx, but, on the contrary, occupy a small depression between the pillars. On section, they show lymphoid tissue with very few crypts (see Figs. XII and XIII). The epithelium growing in from the pharynx appears at times as solid columns, though as a



rule lumen is present. I have observed branching of the crypts only near the pharyngeal surface.

Crypts of adult tonsils as shown by dissection often resemble large pockets, the walls of which may be thrown into folds. A single section through such crypts often give the appearance of branching, caused by sectioning a fold in the wall of the crypt.

Crypts of fetal tonsils may become dilated (See Fig. XIV) and contain keratinous material. Further sectioning of the same paraffin block showed a dilated crypt cut in the transverse plane giving the appearance of cyst formation. (See Fig. XV). Such occurrence does not explain cyst formation in tonsils. Cysts in tonsils are at times sterile and have no demonstrable outlet.

Mucous glands are usually found outside the capsule of the tonsil, and occasionally in the capsule. Mucous glands are found very rarely in the parenchyma of the tonsils (See Fig. XVI). Suppurative inflammation of such glands could give rise to the much disputed tonsillar abscess.

A few of the acini of the mucous glands were much dilated and lined with stratified epithelium. A section through a solitary acini of that description would give the appearance of cyst formation.

#### BIBLIOGRAPHY.

1. Semon and Williams: Allbutt's System of Medicine.
2. Weller: Arch Int. Med., 1921, 27, 631.
3. Mitchell: J. Path. and Bact., 1917, 21, 248.
4. Park and Krumwiede: Jour. Med. Research, 23, 1910, 205.
5. Griffith: Lancet, 1, 1917, 216.
6. MacLachlan: Tonsillitis: Publication from the University of Pittsburgh, 1912.

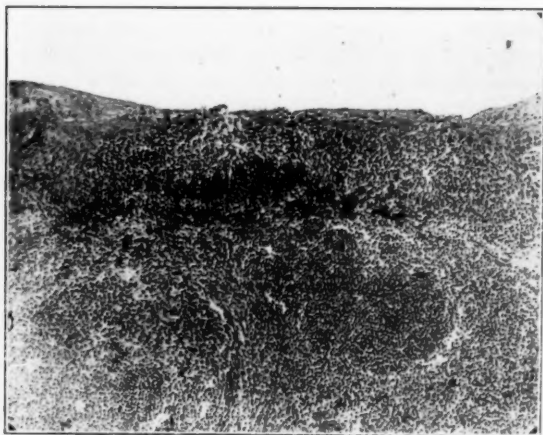


Fig. I. Showing focal thinning of the epithelium lining a crypt and the deep hematoxylin staining material beneath this area.



Fig. II. Showing necrosis of center of a follicle in the tonsil.

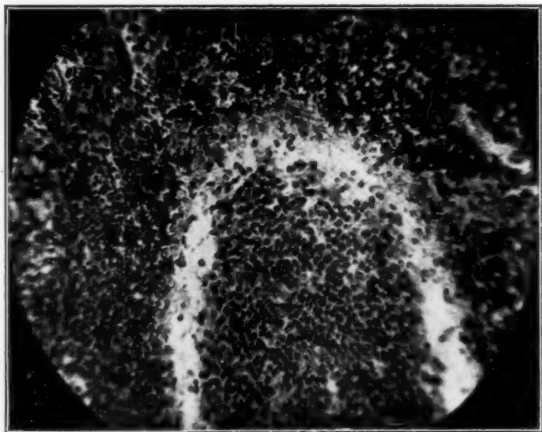


Fig. III. Showing the crescentic arrangement of the deep hematoxylin staining material. Within the "crescent" is a zone of necrosis surrounding the center of the follicle where regeneration is taking place.

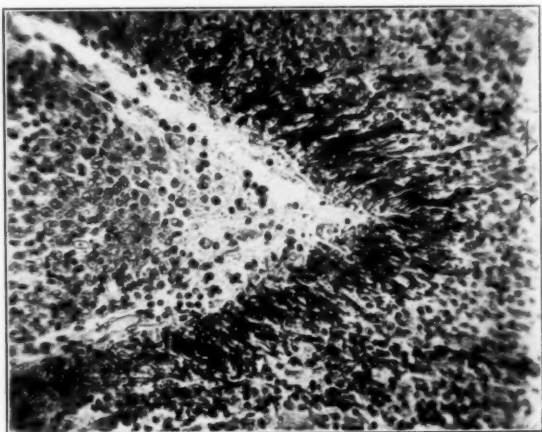


Fig. IV. Showing the radial arrangement occasionally seen of the deep hematoxylin staining material surrounding an area of necrosis at the border of a lymph follicle.

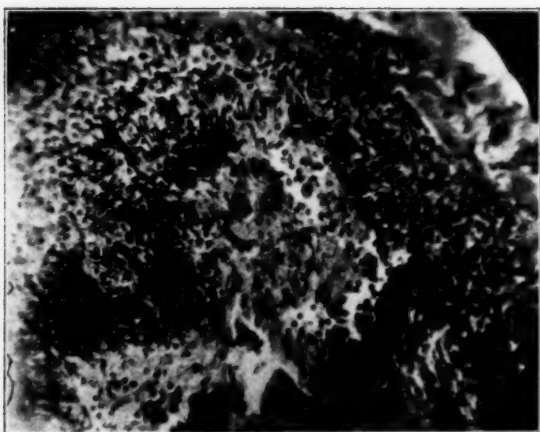


Fig. V. Showing cells with marked pyknosis and karyolysis.  
Many are elongated and slightly club shaped.

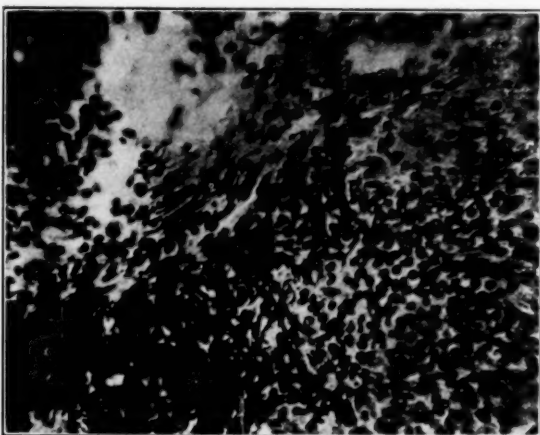


Fig. VI. Showing cell with pyknosis and karyolysis.

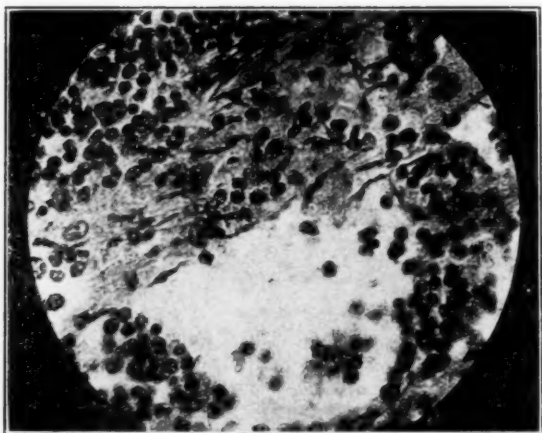


Fig. VII. Showing lymphocytes which are elongated and have a shrunken appearance.



Fig. VIII. Showing a progressive fibrosis. The light staining part represents connective tissue, and the dark fibroblasts the cells of newly forming capillaries, and lymphocytes.

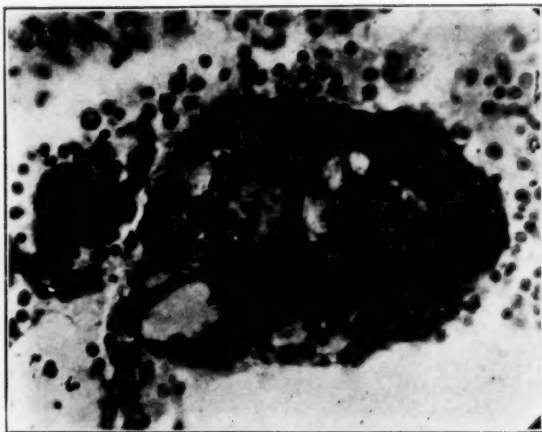


Fig. IX. An "actinomyces-like" granule showing partial calcification. Filamentous organisms and cocci can be recognized in the noncalcified parts. Others in the same section showed complete calcification.



Fig. X. Tonsil from clinical acute tonsillitis showing plugs of fibrinopurulent exudate extending the entire length of the lacunæ.

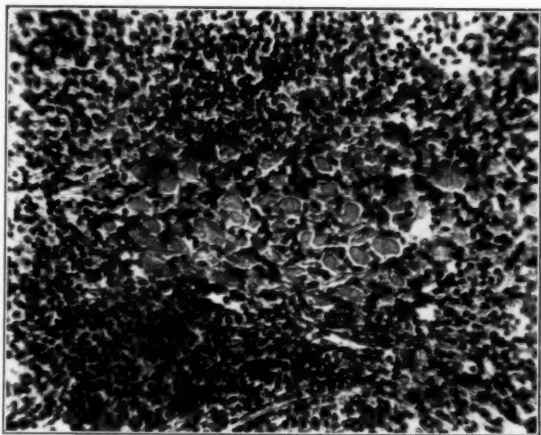


Fig. XI. Transverse section of striated muscle fibers in center of a lymph follicle of the tonsil.



Fig. XII. Tonsil of infant at term showing few lacunæ.



Fig. XIII. Tonsil from infant at term showing branching of lacunæ.

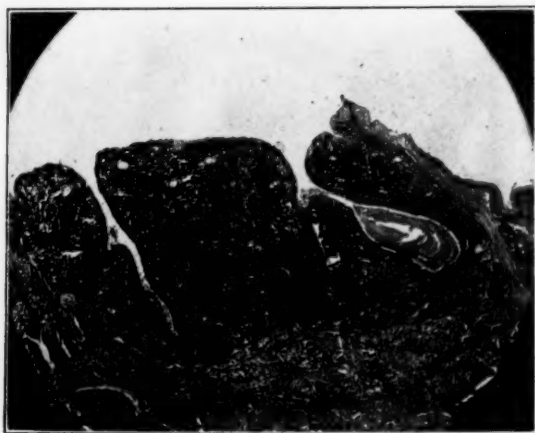


Fig. XIV. Tonsil from infant at term showing dilated crypt filled with keratinous material.



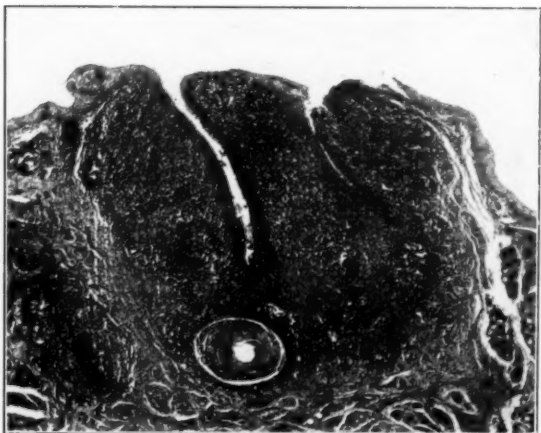


Fig. XV. Tonsil from infant at term showing dilated crypt filled with keratinous material.

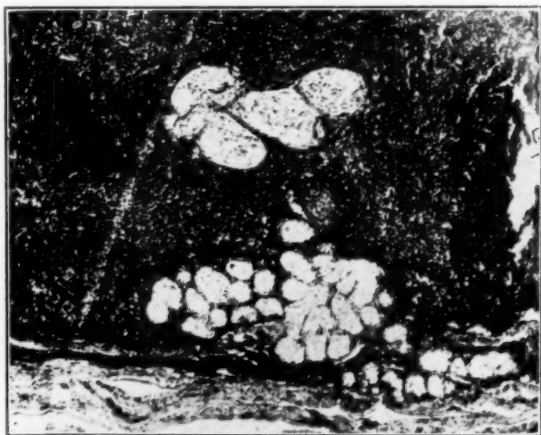


Fig. XVI. Mucous glands in parenchyma of tonsils.

## SARCOMA OF THE ESOPHAGUS.

BY HENRY B. ORTON, M. D.,

NEWARK.

Historical Data.—Bertholet in "The Arch. méd. Exper. Is. 23; 135-150, Paris, 1911," collected 46 cases, reported in the literature between 1875 and 1909. I have collected 4 additional cases and one of my own, which I am herein reporting. Not many varieties of neoplasms and tumors develop in the esophagus, and with the exception of carcinoma they have slight pathologic significance. Those occurring frequently cause no functional disorder, while those occurring occasionally, producing disorders, are so rare that the majority of physicians, as well as the busiest of surgeons, seldom see cases of sarcoma. This statement still holds good and is confirmed by statistics of the Eppendorf Hospital, Hamburg, showing that from 1888 to 1909 no case of sarcoma of the esophagus was observed, while 350 cases of carcinoma of the esophagus were found at autopsy. Professor Chevalier Jackson, with his vast number of esophagoscopies, reports 6 cases, while Da Costa, in the eighth edition of "Modern Surgery," does not mention it. Coplin reports 175 cases of sarcoma of the alimentary tract, collected by Corner and Fairbank, but 14 were esophageal growth. In these cases, I am not reporting those of hypo- or oropharynx, although I append records to complete case.

Definition.—Organic obstruction of the esophagus, due to tumors of esophageal wall, is manifested by slowly increasing dysphagia, which is in turn followed either by regurgitation of the food or dilatation of the esophagus above the point of obstruction. Pain and emaciation are constant; discharge of blood and mucus is common; death being due to starvation or exhaustion.

Causes.—In the resume of these cases it is difficult to ascertain any causes that might predispose to sarcoma, and the writer would not even attempt any suggestions as to them.

In spite of the enormous concentration of effort directed towards its investigation, the nature and origin of sarcoma constitute one of the great unsolved problems of medical science, which thus far has baffled all inquiry.

Whether bad teeth, thereby not properly masticating the food which naturally is swallowed in large boluses, followed by very hot drinks, has any influence as a metabolic or nutritional factor we do not know; it might, however, be found to be one of the common denominators of the final solution of the problem. Curiously, however, all cases of malignant disease of the esophagus which I have seen have had bad teeth.

Race factors, *per se*, do not appear to exercise much influence upon the incident of sarcoma.

Age.—Whatever may be the ultimate nature and origin of cancer it is unquestionably a disease of age.

Heredity.—The relation of heredity to cancer has long been the center of scientific controversy, and it still remains undecided.

The precancerous conditions in the esophagus might be luetic strictures, strictures from injury, caustics, etc.

In these cases no mention was made of the condition of the teeth. Alcohol and tobacco were mentioned (8), and in the case of my own, trauma was attributed by the patient as the beginning of his trouble.

Symptoms.—The symptoms appear in about the following order. Patients have been treated previously for various conditions such as rheumatism, pleurisy, dyspepsia, etc. Suddenly they have noticed difficulty in swallowing food, especially meat, followed by pain in the throat of a burning, stabbing or darting character, maybe referred to the ear, the side, shoulder and down the arm, later, as stenosis increases, they have a regurgitation of food and discharge of a thick, glary, whitish yellow material streaked with blood. Cough is caused by overflow of this discharge into the larynx. As pressure increases, depending upon the location of the growth, difficulty in breathing appears and the vessels of the face and neck may become congested. Ulceration of the esophagus and trachea takes place, causing various pulmonary symptoms. Ulceration may take place through the esophagus into the aorta and cause death due to exhaustion or hemorrhage. Still another

clinical picture of an esophageal sarcoma might be as follows: the disease starts acutely at deglutition, or actual stenosis is preceded by dyspeptic symptoms such as loss of appetite, heart burn, nausea, pressure in cardiac region or behind sternum. Pain commences early and seems to dominate the entire aspect of the disease. It is intermittent with painless periods and occurs usually at night. Periods of disordered deglutition may change off with painless intervals. Stenosis increases, at first only solid food cannot pass and is regurgitated, later fluids cannot pass into the stomach and if death does not intervene the patient is in danger of starving. General health deteriorates with the increasing stenosis and the growth of the tumor. In diagnosing esophageal sarcoma the age of the patient is not of the same significance as in diagnosing other types of sarcoma. For esophageal sarcoma is not by preference a disease of youth. No conclusions can be reached concerning sex frequency from the few cases reported.

#### ANALYSIS OF SYMPTOMS.

Age.—Of the 41 cases reported which gave the age we find that 11 were afflicted with the disease between the ages of 60 and 69, 10 between the ages of 50 and 59, 10 between the ages of 40 and 49, 5 between the ages of 30 and 39, 3 between the ages of 70 and 79, 1 at 80, and 1 at 4, which makes the period between 40 and 69 as that in which sarcoma occurs. There are, however, cases in other decades, the oldest at 80 and the youngest at 4. That sarcoma, therefore, usually occurs late in life and that the period of 40 to 70 years shows the greatest frequency are shown by the above. Age, however, cannot be considered of any diagnostic value.

Sex.—Of the 41 cases reported that give the sex, 31 were males and 10 females, giving a ratio of 3 to 1. More cases of esophageal sarcoma occurs among men in the proportion of 3 to 1, and I think we can exclude that sex as of no significant diagnostic value.

Symptoms.—The first symptom was the fact that they suddenly noticed that while eating some article of food, mostly bread or meat, it had some difficulty in passing to the stomach. In one case (13) it gave a sensation of a foreign body, while in two others (5) and (7) patients first complained of a sore

throat. This was followed by vomiting and regurgitation. In some cases the vomitus would come through the nostrils, the regurgitant material varying from food contents to bloody fluid.

Pain.—All had pain of a varying character, fortunately late in life. Although in some it was intermittent, in others the pain was constant, burning, stabbing and darting in character and was located in the throat, head, neck, shoulder, substernal, back, gastric, thoracic, arms and fingers. In one case (35) the pain was referred to the spinal region. The most common, however, was in the thoracic region. All pain would be increased on swallowing. The difficulty in swallowing increased in all cases; first the inability to swallow solids, then semisolids and finally also liquids, gradually increasing until absolutely nothing would pass, necessitating later either a gastrostomy or rectal feeding, which of course, was only temporary, most all of them dying from inanition.

Difficulty in Swallowing.—This was always noticed. It always increased, some complained of food going only a short distance. In some cases it was accompanied with pain, this beginning at the sternal notch and continuing to the stomach.

Nourishment was gradually diminished, due to inability to swallow. Some cases were fed by rectum, others by gastrostomy, but a very rapid decline in health was noticed as soon as the difficulty in swallowing increased, and unless a gastrostomy was done death has quickly followed.

Laryngeal Symptoms.—Due to an overflow of liquid in the larynx in a number of cases, bloody expectoration when ulceration had taken place, with difficulty in breathing from pressure.

Physical examination of patients was very meager, and in only one was there any history of tobacco. The vast majority of them gave very few symptoms other than of the esophagus.

The Varieties of Sarcoma.—Eighteen were classified simply as sarcoma, 14 as spindle cell sarcoma, 4 as lymphosarcoma, 4 as round cell sarcoma, 2 as fibrosarcoma, 2 as myeloid sarcoma, 2 as melanotic sarcoma, 5 unclassified—that is, no history of case other than mention of the fact that sarcoma existed as in cases 38, 41, 42, 47 and 40.

Location of Growth—Inferior constriction of pharynx, 2; third dorsal vertebrae, 1; at bifurcation of trachea, 5; not mentioned, 18; below cricoid cartilage 5 inches, 15 cm., 4 cm., 3; lower end one-third, 8; below bifurcation, 4 cm., 2 cm., 2; lower end, 12 cm. from stomach, 1; lower end, 10 cm. from stomach, 1; lower end, 1 cm. from stomach, 1; 15 cm. in esophagus, 2; thoracic opening, 1; postcricoid, 4; middle third, 1.

It is worthy of note that in females they were located at middle third or above, most of them at the cricoid, whereas in men they were mostly from the middle third to the end. Also, if located at or near the bifurcation of the trachea, all cases suffered with cough and more laryngeal symptoms with marked difficulty in breathing.

Type: A—Circumscribed, most frequently growing toward the esophageal lumen, the spindle cell sarcoma.

B—The diffuse infiltrating type which tends to metastasis. Both types, however, show tendency to ulceration and degenerative changes.

Metastasis occurs to lymph glands, stomach, intestine, pancreas, kidneys, lungs, pleural cavity, liver, ilium, ribs, cranial bones and brain.

Metastatic growths were noticed in 18 cases, location being in: Lungs, 6; kidney, 1; ribs, 1; brain, 2; pancreas, 1; tongue, 1; femur, 1; stomach, 1; liver, 2; skull, 2.

In diagnosing the esophageal sarcoma one of the primary symptoms is pain. This also occurs in carcinoma, yet, according to Ziemssen, carcinoma may cause prolonged dysphagia, even cachexia, without pain. In sarcoma, however, pain is seldom absent. In carcinoma pain is usually associated with periods of indigestion, while in sarcoma pain is frequently nocturnal or occurs when the stomach is empty. Pain between scapula is most common in carcinoma. It is impossible to differentiate between sarcoma and carcinoma of the esophagus except by esophagoscopy and removal of specimen for microscopic examination.

Prognosis.—Unfavorable. The average duration of life after the onset of disease in sarcoma of the esophagus is five to six months. In carcinoma, seven to eight months, although

in the case of my own it has been twenty-one months since the onset of the symptoms.

**Duration of Life.**—From the onset of symptoms the time was from one to nine months, with the exception of my own case, in which my patient was under treatment for one year nine months and eleven days.

**Treatment.**—Deep X-ray therapy with radium. Gastrostomy palliative. Radical operation unsuccessful as yet. Tracheotomy was done in three; gastrostomy was done in four; subhyoid pharyngotomy in 1. One case disappeared, in which a resection of the esophagus was done. There is no statement of how much of the esophagus was resected.

As to the various operations, palliative or attempted cure, it is very interesting to note that all cases reported, not including my own, which were operated on for tracheotomy and gastrostomy, died within eight days, and of the three cases, not including Dr. Jackson's or my own, in which esophagoscopy was done, all died within eight hours of mediastinitis or rupture. This, of course, was before days of improved technic in passing the esophagoscope.

I now report in detail case 15 and my own case.

**Case 15.**—Hoffman, M. (*Beitr. z. kl. Chir.*, 120, No. 1:201-214, Tueb. 1920), is so interesting that I am reporting it in full.

Subject, male; 61 years old; pain in arms, particularly right arm; thoracic pain; pain at deglutition; increasing emaciation and pain; admitted to hospital—date not given.

**Clinical findings:** No cachexia; weight, 53 kg.; heart and lungs normal; temperature, 37.2, in evening 37.6; esophageal sound No. 12 passes easily into stomach; in extracting it some resistance is felt in the esophagus; a larger sound cannot be introduced for more than 26 cm. in length; obstruction appears to be a black partially slate gray foreign body as large as a filbert; radioscopy, no aneurism; foreign body in the esophagus. Hooked forceps introduced through large esophageal sound; foreign body grasped; in pulling slightly with the forceps a black portion is extirpated as large as a pea.

**Microscopic findings:** Diagnosis of melanotic sarcoma made; teased preparation of glycerin water shows nucleated

tissue, deep black, apparently confirms diagnosis; Thormählen's test negative.

Clinical finding after esophagogastroscopy: Evening temperature, 39.6; over right jugular vein extensive cutaneous emphysema, not very painful to the touch; clear tympanic sound above sternum; distinct crepitation due to attempted extraction.

Operation: Eight hours after esophagogastroscopy, typical gastrostomy in slight ether narcosis, followed by cervical mediastinotomy; slight emphysema to right of thyroid gland; increased emphysema near right jugular vein; two tablespoonfuls of bloody, cloudy fluid drained through esophagus; left side of neck normal; bilateral drain of iodine gauze inserted into mediastinum.

Postoperative findings: Next day, temperature 37.5; pulse 108, strong; night good; tongue dry; medication saline; injection digitalis subcutaneously; feeding through fistula; evening temperature, 38.2; pulse 108; sudden death at night.

Autopsy findings: At base of tongue an ulcerated follicle as large as a pea; 12 cm. above cardia in anterior esophageal wall, two tumors with broad base; first is as large as a pigeon egg, coarsely lobulated, soft greenish gray, smooth surface broken only at the spot corresponding to the upper pole; second tumor is as large as a cherry, very soft, reddish yellow, smooth surface; esophageal mucous membrane is almost entirely infiltrated with white closely adhering strands. Tracheal and bronchial regions infiltrated with a slightly cloudy fluid. Metastasis, to the liver; atrophic, irregular nodulated, surface, infiltrated with broad connective tissue strands; capsule thickened, surface of section blurred, grayish yellow in color. To the spleen: 15 cm. long, 10 cm. broad, capsule thickened, covered with fibroid layer, adherent to adjacent organs, surface rough, color slate gray, surface of section shows pale pink follicles enlarged to size of a millet grain. Death within 48 hours after esophagoscopia from atrophic liver, cirrhosis, chronic splenic tumor, perisplenitis, esophageal polyps, traumatic mediastinal emphysema, fibrous pleuritis.

Microscopic findings: Esophageal polypoid tumor is a richly vascular spindle cell sarcoma; large spindle cells, slightly polymorphous, occasional polynuclear elements, abundant in-



tracelluar pigment, according to Verokay's method, "show here and there scant pigment with golden nuclei among the sarcoma cells—in spite of negative iron reaction the tumor might be considered a hematogenous growth from its shape, color and luster." The surface of section shows a uniformly broad, necrotic zone covering hyperemic tumor cells; occasionally there is some limited leucocytic infiltration instead of the superficial necrosis.

My own case was that of a man, C. A. C., referred to me by Dr. Chas. A. Teeter, of Newark, N. J., on November 9, 1921, for difficulty in swallowing. His age was 47 years, male, white, married, weight 152 pounds, occupation that of a repairman in the Fire Department. His past history showed that he had had measles, mumps, whooping cough and scarlet fever in childhood, had had no operations and has enjoyed good health up until the present trouble. Family history is unimportant; his mother died of some liver trouble; his father died at the age of 85, of some intestinal trouble, the exact cause not being known. He has one brother living and well and one brother dead, cause of death being apoplexy. His wife and two children are living and well.

His present trouble began with pains in the chest about three months prior to his first visit to me. He then had some interference with swallowing. Meat or any hard substance would cause pain in his right breast. He consulted a physician for the pain in his chest, who diagnosed and treated him for pleurisy. He then saw Dr. Teeter, who ordered X-rays made and referred the patient to me for esophagoscopy.

The patient at this time was only able to eat soft diet, as milk, cereals, etc. He had pain across his right chest, radiating to his right arm, had vomited once and had lost some weight, but not recently.

On examination, I found a septum deviated to the right, his middle turbinate in contact with the septum, a number of bad teeth, his pharynx granular, his uvula was reddened, as well as his left vocal cord, which also showed a paralysis. There was a slight amount of mucus in the left pyriform sinus.

On November 12, 1921, at the Presbyterian Hospital, without anesthesia, a 7 mm. full lumen esophagoscope was passed.

At 23 cm. from the teeth a bleeding mass was seen; at 26 cm. from the teeth, granulations on the posterior wall of the esophagus. From there the tube passed readily to the stomach, the time of operation being four minutes. A diagnosis of carcinoma of the esophagus was made. No specimen removed at that time for microscopic examination. On November 14, 1921, a Wassermann was made and reported back negative. At that time the patient complained of a slight fetid odor on belching. Notwithstanding the negative Wassermann, a therapeutic test for specific infection was given. I saw the patient again on November 25, 1921, at which time he said that he felt very well. He did notice that when eating vinegar on salads the acid caused some burning in the region of the growth. The pain radiating to the right arm and chest had disappeared; his therapeutic test still continuing. On December 2, 1921, he had no pain, but complained of a sensation of burning in the esophagus. An esophagoscopy was again suggested, and on December 6, 1921, at the Presbyterian Hospital, without anesthesia, a 7 mm. esophagoscope was passed, and at 23 cm. from the teeth was a sloughing mass. A piece was taken for examination. I was still of the opinion that the patient suffered from carcinoma of the esophagus, notwithstanding the fact that the microscopic examination of the tissue removed was reported back bloodclot, necrotic material and purulent infiltration, the pathologist giving the diagnosis of inflammatory tissue. On December 13, 1921, the patient informed me that at times he had pain in his right breast and seemed to feel that he swallowed a little better, and also made the remark that if he could swallow all food he would not think that there was anything wrong with him. His diet at that time consisted of bread, milk, eggs and coffee. On December 20th, his swallowing was much easier; he was eating thicker substances; had some pain in his right nipple and his weight was 140 pounds; he had lost four pounds in six weeks, his diet consisting of soft boiled eggs, vegetable soup, oysters, chicken broth and mince pie. On December 27th, his condition was about the same. On January 5th, 1922, a 7 mm. esophagoscope was passed, and at 23 cm. from the teeth a sloughing mass was found, a large piece of which was taken for examination. I was still of the

opinion that the mass was carcinoma. I did notice, however, that on examination I encountered more hemorrhage than at any previous examination.

The microscopic examination made by Dr. Gray, at the Presbyterian Hospital, gave the following description of the specimen removed: Firm, white, numerous mononuclear and multinuclear giant cells, large polyhedral cells, round cells and spindle cells. There is a marked activity in the form of mitosis. Diagnosis: Sarcoma of the esophagus.

On January 13, 1922, the patient was eating finely chopped meat and had gained one-half pound. On February 7, 1922, 25 milligrams of radium were placed on the esophagus through the esophagoscope and allowed to remain there at 29 centimeters from the teeth for six and one-half hours. Five days after application of radium he had some pain, which lasted for three days. He was at the same time receiving deep X-ray therapy by Dr. Reissman. On February 28, 1922, 25 milligrams of radium were applied to the esophagus, by means of the esophagoscope, and allowed to remain there at 29 centimeters from the teeth for six and one-half hours. On March 10th, an X-ray was taken, which showed some improvement. He continued to have X-ray treatments, and on March 28th said that he felt better than he had in the last six months. On April 7th, 25 milligrams of radium were applied in the same manner as before for six and one-half hours. The therapeutic test for specific infection was stopped. On April 17th, I noticed some browning of the skin from the X-ray treatment. He complained of some pain down the right arm. On May 15th, the patient had gained one pound. The case was again esophagoscoped on the 29th of May, 1922, and the therapeutic examination of the specimen removed showed the same picture as on January 5th.

By June 12th, he had gained two pounds in weight. Radium, 25 milligrams, was again applied esophagoscopically for six hours. One week later he complained of pain in the midsternal region and soreness in the right side. His X-ray treatments still continued, and on July 27th a roentgenographic reexamination of the esophagus by Dr. Reissman showed no signs of improvement; if anything, the extent of the invasion was greater. He complained for the first time of cough on July 31st.

On August 7th he had some tenderness in the midsternal region, had vomited twice, a pinkish vomitus, and had some difficulty in drinking milk. He continued to go downhill until November 12th, when a gastrostomy was done by Dr. Hawkes. No further X-ray treatment or radium applications were made, the patient dying March 20, 1923. I was anxious to obtain a postmortem, but the family would not give me their consent.

The following is the report of all specimens removed by Dr. Gray: The examination of small pieces of tissue from esophagus of Mr. C. A. C., January 5, 1922, showed nothing in the gross of diagnostic importance—small pieces of firm and soft white tissue. Microscopically numerous giant and polyhedral cells with large hyperchromatic were present. Many of the giant cells were multinuclear and mitotic. They were true giant cells rather than foreign body giant cells, either myoblastic or fibroblastic in origin. May 29, 1922, small pieces of soft tissue from same case show the same, except with a great deal of pus and necrotic material.

October 21, 1922, many large degenerated cells embedded in pus and necrotic material. Diagnosis: Sarcoma of the esophagus.

December 6, 1921. Blood clot and necrotic material. Pus cells. They appear as ulcerative process or as a polypoid tumor, the former containing round or polyhedral cells, the latter spindle cells. Glinsky observed mixed tumors of the esophagus composed of striated muscle, spindle and giant cells. The ulcerative polyhedral cell type is more likely to metastases than the spindle cell type.

#### CONCLUSIONS.

1. Age.—Sarcoma occurs late in life, generally at the period of 41 to 70, although in females it occurs at a much younger age. Age, however, is of no significant diagnostic value.
2. Sex.—About 75 per cent of all cases of sarcoma of the esophagus occur in men, although sex has no significant diagnostic value.
3. Location.—The thoracic portion of the esophagus is most frequently the starting point. Sarcoma shows less preference for the narrow portions of the esophagus than does carcinoma.

4. Type.—(a) Circumscribed, sometimes polypus tumor, most frequently toward the esophageal lumen. (b) Diffuse infiltrating tumor, tends to metastasis.

5. Metastasis.—To almost all organs.

6. Diagnosis can be confirmed only by esophagoscopy, and histologic examination.

7. It is highly important to determine the malignancy of esophageal polypus, sinus both carcinoma and sarcoma of the esophagus may present themselves as genuine polypus neoplasms.

8. Clinical symptoms vary and are unreliable for assuring diagnosis, unless esophagoscopy and removal of specimen.

9. Death usually due to cachexia.

10. Treatment.—Surgically unsatisfactory so far, although Morelli reports removal of benign growth. Radium and deep X-ray therapy seem to be the only means of relieving pain and slight prolongation of life.

#### BIBLIOGRAPHY.

1. Chapman, S. H.: *Am. J. M. Sc.*, 74:433-437, Oct., 1877.
2. Oppenheimer, L. S.: *Louisville Med. News*, 7:74-75, 1897.
3. Shaw, L.: *Trans. Path. Soc., Lond.*, 42:90-91, 1891.
4. Rolleston, H. D.: *Brit. M. J.*, 1:292, Feb. 11, 1893.
5. Paget, S.: *Trans. Path. Soc., Lond.*, 46:44-45, 1895.
6. Osler, C.: *Trans. Path. Soc., Lond.*, 47:40, 1896.
7. James, G. B.: *Tr. Path. Soc., Lond.*, 49:91, 1898.
8. Livingood, L. E.: *Johns Hopk. Hosp.*, 9:159, July, 1898.
9. Howard, W. T.: *J. A. M. A.*, 38:392-399, Feb. 8, 1902.
10. Oppenheimer, L. S.: *Louisville Med. News*, p. 74, 1879.
11. Schlagenhauser: *Virchow's Arch.*, 164, 1901.
12. Kraus: *Diseases of the Oral Cavity and the Esophagus*, 1902.
13. Rosenbach: *Berl. kl. W.*, No. 38, 1875.
14. Eicken: *Deut. Ztschr. f. Chir.*, 65, 1902.
15. Howard: *J. A. M. A.*, Feb., 1902.
16. Hoffman, M.: *Beit. z. kl. Chir.*, 120, No. 1, 201-214; Tüb., 1920.
17. Komer, I.: *Diss.*, Berlin, 1884.
18. Kundrat: *Wien kl. W.*, No. 12, 1893.
19. Rotschy, I.: *Diss.*, Geneva, 1897.
20. James: *Tr. Path. Soc.*, 47:40, Lond., 1898.
21. Reutter: *Trans. Inst. Path. de Lausanne*, No. 3, 1919, 1901.
22. Von Hausemann: *Microscopic Diagnosis of Malignant Tumors*, p. 71, Berl., 1902.
23. Frangenheim, V. A.: 164:147, 1906.
24. Joliat: *Thèse*, Lausanne, 1907.
25. Bertholet: *Arch. Med. Exper. Is.*, 23:135-150; Paris, 1911.
26. Dubreuil: *Gaz. med. de Paris*, No. 24, 1885.
27. Targett: *Tr. Pathol. Soc.*, 40, Lond., 1889.

27. Stephan: Jhrb. f. Kinderhkl., 30, 1890.
28. Shaw, L.: Tr. Path. Soc., 42, Lond., 1891.
29. Wolfensberger: Zeigler's Beitr., 15, 1894.
30. Albrecht: Wien kl. W., No. 18, 1895.
31. Von Northaft: Muench. m. W., No. 15, 1895.
32. Brooksbank: Tr. Path. Soc., 49, Lond., 1898.
33. Gastpar: Ztbl. f. allg. Path. and Path. Anat., 11, Nos. 3 and 4, 1900.
34. Stark: Virch. Arch., 162, 1900.
- 35.
36. Glinski: Virchow's Arch., 167, 1902.
37. Von Eicken: Deut. Ztsch. f. Chir., 65, 1902.
38. Percy: Siglo med., 1, 1903.
39. Wegener, I.: Dissert., Giessen, 1904.
40. Frattin: Policlin. ser. chir., X. G., 11, 1903.
41. Baur: Arb. a. d. path. Inst., Tübingen, 5, No. 2, 1905.
42. Donath: Virchow's Arch., 191, No. 3, 1908.
- 43.
44. Borrmann: Verh. de Deut. Path. Ges., Kiel, Apr., 1908.
45. Herxheimer: Verh. de Deut. Path. Ges., Kiel, Apr., 1908.
46. Von Hacker: Mitteil. a. d. Grenzgeb. d. Med. and Chir., 19, No. 3, 1908.
- 47.
48. Rieke: Virchow's Arch., 198, No. 4, 526-545, Bul. Dec. 4, 1909.
49. Kolmer, J. A. and R. S. McCombs: Proc. Path. Soc., Phila., 35:16-17, 1915.
50. Jackson, Chevalier: Personal communication.
51. Orton, H. B.: Sarcoma of the Esophagus.
52. DaCosta: Modern Surgery.
53. Coplin: Manual of Pathology.

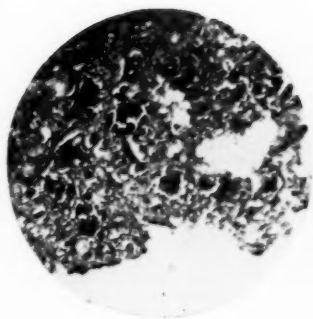


Fig. 1.  
Enormous cells; mitotic, small  
round lymphocytes.

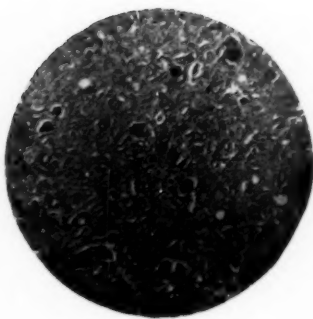


Fig. 2.  
Large mitotic cells.



Fig. 3.  
Sarcoma. Composed of tumor  
and blood vessels, in upper  
right space.

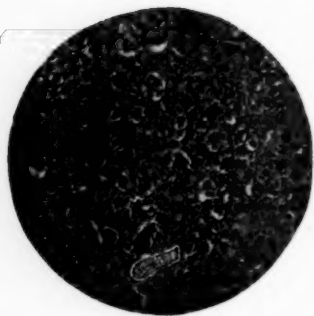


Fig. 4.  
Sarcoma. Numerous giant cells.



Fig. 5.  
Low power, showing tumors and  
other cells.



NASAL HUMP—A SIMPLIFIED EXTERNAL  
OPERATION.

SAM E. ROBERTS, M. D., F. A. C. S.,

ASSISTANT PROFESSOR RHINOLOGY, UNIVERSITY OF KANSAS,

KANSAS CITY.

External nasal deformities, from whatever source, or whatever type, are a constant source of annoyance to the patient. It produces a mental state which is difficult for the casual observer to understand. It is only when they unburden to their physician, in seeking relief, that the whole story is told. Self consciousness, reticence and at times even a melancholia follow. The surgeon must differentiate between the ones actually needing help, whose business and social life is disturbed, and those whose deformity is only slight and needs no correction.

Nasal hump is by far the most common external deformity. There are two distinct types, congenital aquiline and traumatic. The congenital is the more common, is a racial and familial characteristic. It may vary from a slight elevation of the bridge with the nasal tip not disturbed, to a huge, broad base, large hump, tip pushed down hook shaped like an eagle's beak. Certain families have a characteristic hump. The true aquiline nose is most often found among the Semitic peoples. The traumatic hump is an exostosis due to a bony proliferation from an injured periosteum. Carter,<sup>1</sup> in discussing the dynamics of nasal development, states "The pressure exerted by the septum during the period of growth is in an upward and forward formation, with little or no force horizontally in the direction of the tip. . . . Vomer and arch of the hard palate are not yielding in the vertical plane downward, hence upward growth is the only direction possible." He also advances the ingenious and logical theory that hump may be prevented in children of a family where it is a characteristic by high septal resection in early adolescence.

The treatment of nasal hump is entirely surgical. The two methods of approach are intranasal and extranasal. The literature of the last five years is almost entirely devoted to the intranasal method, but in my own experience I have found the extranasal route, by the method I will describe, much more satisfactory.

The nose is irrigated with warm sterile normal saline solution several times the day preceding and one-half hour before the operation. The entire face is washed with green soap and water just before the anesthetic (gas ether) is started. After the patient is anesthetized, cotton swabs of tincture of iodine are mopped in the vestibule of the nose. The nasal cavity is then packed with strips of gauze dipped in 2 per cent iodine. The anesthesia is continued entirely through the mouth. All exposed skin surface is painted with 2 per cent iodine.

A central incision (as shown in Fig. 1) is made underneath the tip of the nose.

The incision is started in the center with a stab and then widened on each side. Scissors are inserted (Fig. 2), spread apart, then advanced slowly upward with points down toward the periosteum, making only short cuts and each time being particularly careful that neither the skin nor the intranasal mucosa is punctured. After the elevation is completed an Alexander gouge is inserted, and the lower part of the hump engaged (Fig. 3); with a few taps of the mallet the hump can be smoothly cut from its base.

The removal of the bone (Fig. 4) I have found the most difficult step and requires the most patience.

All blood is expressed from the wound, and while an assistant holds pressure over the bridge the wound is entirely closed with silk gut. A pressure dressing over the bridge is applied and held in position firmly with adhesive.

I believe there are three distinct advantages of this technic over the intranasal method.

First.—A more aseptic field.

Second.—Greater accessibility and visibility.

Third.—A smooth cut is made, leaving no bony irregularities which have to be rasped away.

The objections to the external methods of operating have always been the scar. This objection is real, where a longitudinal incision over the hump is made, but it certainly does not apply where the incision is made underneath the tip. All that can be seen a few weeks after operation is a thin white line when the patient's head is tipped slightly backward.

## BIBLIOGRAPHY.

1. Carter: Medical Record, May 22, 1920.  
906-7 WALDHEIM BLDG.

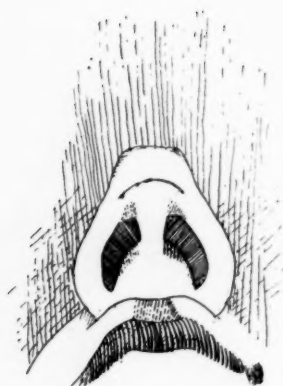


Fig. 1.

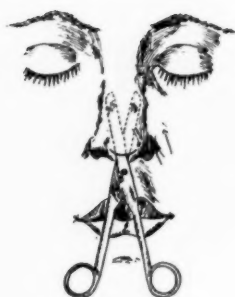


Fig. 2.



Fig. 3.



Fig. 4.



Fig. 5.



Fig. 6.  
Six weeks after operation.



Fig. 7.  
Before operation.



Fig. 8.  
Seven weeks after operation.

## THE PERSISTENT INFANTILE ETHMOID.

BY JOHN J. SHEA, M. D.,

MEMPHIS.

Wittmaack, of the University of Jena, published in 1918 two volumes, "Über die normale und die pathologische Pneumatisation des Schläfenbeines," and "Einschließlich ihrer Beziehungen zu den Mittelohrerkrankungen," one of which is a text and the other an atlas. He described the persistent infantile mastoid and gives as its cause infections of the bone early in childhood. Infections of the middle ear limit the growth of the mastoid bone and its pneumatization is retarded. The prototype of the persistent infantile mastoid is seen in the persistent infantile ethmoid. Unfortunately, the author has not had the advantage of many autopsies on the ethmoids of infants, but has drawn the material for this paper from operative cases.

The ethmoid cells are present at birth. They develop until puberty is well established and reflect in their growth the influences of the infections they have suffered.<sup>1</sup> The sphenoid sinus is generally a distinct bony cell at birth, but has no clinical significance. From the age of three the sphenoid sinus grows rapidly, so that at the age of ten it reaches its adult size.<sup>2</sup> The frontal sinus is formed by the migration of an anterior ethmoid cell into the frontal bone, and as soon as the cell enters the frontal bone it becomes the frontal sinus.<sup>3</sup> At birth and during the early years of life the ethmoid cells are sharply divided into an anterior group and a posterior group. The sphenoeethmoid labyrinth will assume the size and shape that its component bones will allow, and the presence or shape of the frontal sinus is governed by the migration of the anterior ethmoid cells. The growth of the sphenoeethmoid labyrinth should be proportionately pneumatization and capsulation. In the persistent infantile sphenoeethmoid labyrinth the pneumatization is subnormal and the capsule hypertrophied, as

shown by the roentgenogram. The characteristic persistent infantile sphenoethmoid labyrinth in the adult has few anterior cells and these are small with hard, thick walls which do not migrate sufficiently to form a frontal sinus, or if they do form a frontal sinus, it is only rudimentary. The posterior cells are likewise few with hard, thick walls, but are larger than the average posterior cell, though their aggregate volume is less. The sphenoid sinus is small and its walls are thick and firm. The membrane lining these cells is thin and not capable of normal secretory powers.

The role of the endocrines is speculative, but evidently a potent factor in many of these cases. Some of our adults, possessing persistent infantile ethmoid, could be classed as subnormal endocrine subjects, whereas others were average individuals, and one (Case 1) was of the hyperpituitary class, as proven by his massive build and a large sella turcica shown by the roentgenogram.

Dean,<sup>4</sup> in a recent talk before the laryngologic section of the Academy of Medicine in New York City, cites feeding experiments whereby certain diets would produce a suppurative paranasal sinus disease when fed to white rats. It has been our experience that a diet high in carbohydrates will predispose infants to acute infections of the upper respiratory tract.

The infections in the children may be from any of the simple suppurative bacteria or the virus of syphilis. Some of the cases have a continuous infection from infancy to adult life, while others have an interval between the ages of four to six years and early adult life. In this latter group the infecting organisms have varied through the entire simple suppurative varieties. The clinical course in the adult with persistent infantile ethmoids differs from that in the normal adult in that there is greater pain, apparently due to the osteitis, and its course is more chronic. When first seen in the adult, these cases may give the appearance of the so-called atrophic rhinitis because the turbinates are small and the discharge chronic. The lack of sufficient moisture more readily dries the discharge into scabs and crusts. The roentgenogram will tell the story. Here will be found a skull with absent or rudimentary frontal sinuses; small ethmoids with thick walls, cloudy or cystic; small sphenoid sinuses with heavy walls and small antra.

The clinical course is towards chronicity and, even though operated upon, the healing is slow. This is very notable where on one side the sphenoid is of average size and on the other rudimentary. The normal sphenoid will follow the natural convalescence, while the rudimentary sphenoid will not heal readily and requires long and persistent care.

The treatment in the adult is, as a rule, surgical and requires prolonged and careful postoperative care. Of course the best treatment would be the prevention of the persistent infantile sinus by the early recognition in the infant and the institution of the necessary steps to cure or arrest these infections in early life.

#### CASE HISTORIES.

Case 1.—P. T., age 12, with a history of infection of sinuses beginning during the first year of life and persisting until the present. The examination showed an atrophic nose, small middle turbinates, and many scabs and crusts. The tonsils and adenoids have been removed. The roentgenogram shows the absence of the frontal sinus, which substantiates the history of the infection beginning the first year of life. The sella turcica was large. The ethmoids have the characteristic hard, thick walls, with one large posterior cell. The left antrum is cloudy. The findings at operation were: The middle turbinates were atrophic; the ethmoids, anterior group, firm and few cells, the posterior group was of the large cell type and contained mucopurulent discharge; the sphenoid was large and easily entered and its membrane was normal; the left antrum was filled with a coagulated mucopurulent mass, and the lining membrane was thickened. The massive build and large sella turcica were indications of a hyperpituitarism. This case illustrates three facts: (a) That infections beginning during the first year of life will retard the pneumatization of the sinuses and produce persistent infantile ethmoids and an absence of frontal sinuses; (b) that in these persistent infantile sinuses the inflammations assume atrophic characteristics; (c) that hyperpituitarism can be present as well as hypopituitarism.

Case 2.—Miss M. C., age 28, chief complaint was pain in and about left ear, for which her tonsils had been removed



and local nasal treatment given a lengthy trial with no results. Examination of the ears failed to reveal any deafness and inspection of both membrana tympani was normal. The membrane of the nose was thin and dry. The left middle turbinate was of average size, but free of any contact. The septum was badly deflected to the right, imposing on the right middle turbinate, the latter being atrophic. Transillumination failed to throw any light upon the pathology. The roentgenogram showed on the right side persistent infantile ethmoids and a rudimentary frontal sinus. The cells on the left side were well developed with a cystic posterior ethmoid cell. At operation there was found a large spur to the right formed by the vomer, as if an injury had been sustained at an early age. This deformity evidently was the cause of an arrest of development on the right side. The membrane of the right cells was atrophic and that of the left side was polypoid. This case illustrates (a) that accidents may retard the pneumatization of the ethmoid cells, and the sphenoethmoid labyrinth is limited in its development by its component bones; (b) that the same infection that will produce hypertrophic forms of inflammations in the normally developed cells will produce atrophic changes in the persistent infantile sphenoethmoid labyrinth.

Case 3.—Mrs. B., age 33. Roentgenogram of mastoid shows persistent infantile mastoid and roentgenogram of sinuses shows rudimentary frontals. At operation the right mastoid was of the sclerotic type with especially few cells. Later it became necessary to exenterate the ethmoids, and they were found to be of the persistent infantile type and also demonstrated the tendency towards atypically placed cells. Fortunately I was able to enter and drain the cell shown above the left orbit, which is below what should have been the floor of the left frontal sinus. This was a true supraorbital ethmoid cell and evidently an offshoot from one of the anterior group, whose migration should have been into the frontal bone to have formed the frontal sinus. This case illustrates how the mastoid process and the nasal sinuses can both be of the persistent infantile type and suggests some endocrinal disturbance. This particular patient was a female, 33 years of age, with many of the features of a status lymphaticus subject. She was extremely blonde, fair skinned, with excessive subdermal fat.

## REFERENCES.

1. Dean, Lee Wallace: "Paranasal Sinus Diseases in Infants and Young Children." University of Iowa, Studies in Medicine, April 15, 1921.
2. Schaeffer, J. Parson: "The Nose and Olfactory Organs." P. Blakiston's Son & Company, Philadelphia, Pa., page 178.
3. Schaeffer, J. Parson: "The Nose and Olfactory Organs." P.
4. Dean, Lee Wallace: Iowa City, Iowa. Personal communication, Blakiston's Son & Company, Philadelphia, Pa., page 143.



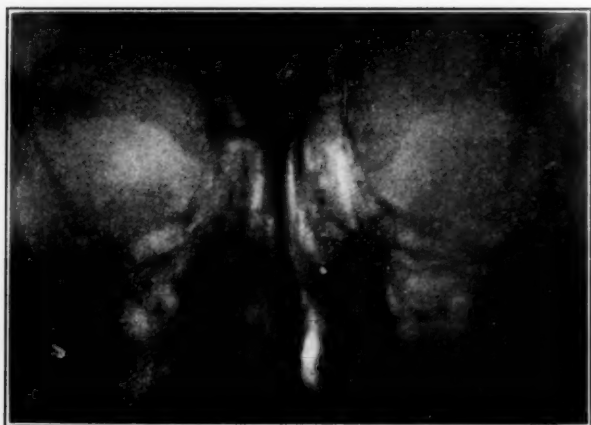
Case 1.

The roentgenogram shows the absence of the frontal sinus. The ethmoids have the characteristic hard, thick walls, with one large posterior cell. The left antrum is cloudy.



Case 2.

The roentgenogram showed on the right side persistent infantile ethmoids and a rudimentary frontal sinus. The cells on the left side were well developed with a cystic posterior ethmoid cell.



Case 3.  
Roentgenogram of sinuses shows rudimentary frontals.



Case 4.

#### LXXIV.

### ATROPHIC RHINITIS AND OZENA—HALLE OPERATION.

BY A. J. LORIE, M. D., AND PAUL LUX, M. D.,

KANSAS CITY.

Although not an uncommon disease, ozena has been relegated so strictly to the sphere of the rhinologists that but little is known of it outside of this specialty.

The term ozena comes from the Greek and means, literally, a stench. This condition has been known since antiquity and was thought by the ancients to have been due to an excessive secretion from the brain into the nose. Literature is full of methods and means of treating this condition, all of which in the past have proved only moderately or not at all successful. Every method of which the ingenuity of man might conceive has been tried.

Baumgartner and Chiari have called attention to a family tendency to this condition. Among 46,000 patients coming to the clinic for all manner of ear, nose and throat conditions, 880 of these patients had ozena; 357 were male, 523 female.

The etiology of the disease has been a question of dispute for a number of years. Perez claimed to have found a coccobacillus for which a specific connection with the disease was established. The coccobacillus of Perez, this organism in culture, gives off the characteristic odor of this sweetish, sickening stench which so characterizes ozena.

Hofer and Kofler, working together with this organism, treated numerous patients with an autogenous vaccin of Perez, and they claimed to have obtained considerable improvement. This article, when published in 1917, caused many men to follow their lead. One of us (Lorie) was among those who attempted work along this line, isolating the organism and making the vaccin at the Research Hospital laboratories and treating many patients without any appreciable result.

There are now two schools, one following the views of Lautenschlager and Grünwald, who contend that this disease

originates in the nasal sinuses in childhood, and that the pathologic changes occur in the bones and mucous membrane; the atrophy and crusting are secondary in importance. This idea is shared by numerous American rhinologists. They claim that the ethmoid and frontal sinuses are rarely responsible for this condition.

On the other hand, there is a group of men who follow the teachings of Max Halle, of Berlin, who questions the correctness of the above theory, as it fails to explain why a minority of the antrum infections of children lead to ozena, while a majority develop into a typical empyema, such as that described by Dean of Iowa City. We know that ozena is usually bilateral, while this is not true of empyema, and that this disease is more frequently found among the children of the poor. We know that from the viewpoint of Halle, the best results obtained in the treatment of the disease have been and are being obtained by following the latter school. As to the etiology of ozena, while these two schools differ in the main as to the causation of ozena, they are almost uniform in their method of treatment, with the exception of the procedure.

The pathology of ozena and atrophic rhinitis has been rather definitely worked out. Axisa, of Naples, 1922, observed the evolution of ozena. First symptom was an abundant grayish discharge without odor. This seemed to be coming from the ethmoids and mucous membrane surrounding them. Secondly came the odor, then crust formation; first on the middle turbinate and not adherent because of the abundant secretion, while later the crusts increased and the secretion decreased.

Herman Sternberg,<sup>1</sup> Berlin, 1923, published a rather classical article upon the pathology of ozena. He examined histologically pieces of mucous membrane and bone from the middle and inferior turbinates of fifteen cases of ozena. He has found, with others, that the changes are from the ciliated normal epithelium to the squamous epithelium, with infiltration of the mucosa progressing to a transition into fibrous connective tissue, with a shrinkage of the mucosa; a disappearance of the glands which normally lodge in the turbinates and an atrophy of the bone following an initial rarefying osteitis with pigmentation and endarteritis of the small arteries. He disputes only the accepted view of the previous workers, of the non-

specific character of the infiltration. He says the infiltrate differs from other infiltrates of an inflammatory character of the nasal mucosa by the frequency of hyalin degeneration with the so-called Russell bodies and by the abundance of mast cells as they are usually found in chronic specific inflammatory granulations, such as actinomycosis and rhinoscleroma, and particularly in chronic inflammatory diseases leading to atrophy, such as gastritis atrophica. Thus the ozena infiltrate is considered analogous to that of atrophic gastritis.

The metaplasia of the ciliated epithelium is only a consequence of the chronic inflammation of the mucous membrane. This metaplasia cannot be explained solely by the secretions and crusts in ozena and chronic suppuration of the sinuses. The question naturally arises whether the absence of leucocytes passing through the epithelium and the lack of their liquefying ferment is partly responsible for the crust formation.

Caldera,<sup>2</sup> May, 1922, in Munich, attempted to prove the neurotrophic changes in atrophic rhinitis are primary causes. He resected the maxillary nerve of rabbits and then injected the specific so-called Perez organism of ozena into the sinuses, but no ozena developed.

We differentiate atrophic rhinitis and ozena simply in degree. By both we mean a chronic disease process of the nasal structures, accompanied by nasal atrophy of both the nasal mucosa and the underlying bony structure. There is a secretion thrown off in a more or less degree. If this secretion is characterized by stench, it is designated as ozena; if there is no odor, we call it a simple atrophic rhinitis. Histologically we found a small cell infiltration of the new growth of connective tissue with an obliteration of the small blood vessels and an endarteritis. A change from the characteristic ciliated columnar epithelium to a squamous type. This will later yield to a pressure atrophy of the underlying periosteum. This pressure atrophy is further increased by the lack of nutrition due to the blockage of these small vessels.

The most characteristic symptoms are the crusting and secretion. The secretion consists of tissue detritus and is further supplied with fluid; it is of a tenacious type and has a remarkable tendency to drying and crusting. These crusts take on several characteristics, they may be yellow, gray or black.

They may be so extensive that they form a complete cast over the interior of the nose, so that these patients complain of difficult nasal respiration due to a complete blockage of their nose. We usually find a small amount of secretion under this crusting which does not have the characteristic ozena odor.

The diagnosis is easy; it depends upon the absence of ulceration, the absence of bone necrosis and the presence of scabs and crusting; the atrophied appearance and shrinkage of the mucous membrane and the sickening sweetish odor, together with the gelatinous appearance of the mucous membrane. One of course must realize that there are periods when the odor of ozena disappears. At this time ozena is difficult to differentiate from atrophic rhinitis.

Inspection of the nose shows that the lateral wall, septum and floor are covered with crusts, which very frequently cover the nasal pharyngeal wall. The nose is wide and roomy. Upon removing these crusts, we find that there is a marked atrophy of the middle and inferior turbinates, and in extreme cases there is almost an absence of inferior turbinate. In extreme cases one can obtain an excellent view of the eustachian tube and even the sphenoid opening.

These patients are in a more or less pitiable condition. They are in a sense social lepers. They have such a terrific odor emanating from their nares that it is sometimes possible to diagnose ozena by the stench that comes into a room with the patient's presence.

In the past we have been prone to tell these patients that they have a hopeless condition and to condemn them to a life of nasal douches, nasal massage, with more or less social ostracism. There have been cases reported of marriage separations based upon this disease. (Beck.) We claim there is not one of these patients who cannot be helped to such a remarkable degree that they are more or less free from these distressing symptoms for the rest of their lives.

Among the numerous treatments that have been discarded we can briefly enumerate: First, that of paraffin injection under the lateral wall of the inferior turbinate; secondly, pieces of tibia which have been inserted under the mucous membrane of the septum, and ivory, bone and fat transplants.

Fleischman,<sup>3</sup> 1922, in Berlin, described the bacillus of Perez



as a harmless organism and the vaccin therapy as simply an irritating measure. Bruno Bruzzi<sup>4</sup> treated his patients by insufflation of lactic acid. Kowen of London insufflates oxygen.

Lautenschlager, 1917, before the Berlin Laryngological Society, published his treatment by means of his operation for atrophic rhinitis. This, in my opinion, is the greatest single advance that has been made upon this subject.

Lautenschlager<sup>5</sup> has obtained good results in the treatment of these cases by an operative procedure in which he advances the lateral nasal wall toward the middle line and removes the antral lining. In order to obtain a healthy lining of the antrum he inverts a large portion of oral mucous membrane into the cavity and packs for as long a time as possible. More recently he has advocated the inversion of Stenson's duct, so that the resulting continuous flow of saliva may afford a further stimulus to the bone. As the opening from the antrum into the mouth is being maintained in this way, the saliva ultimately reaches the latter cavity.

Lautenschlager's operation is a rather formidable procedure, requiring a rather mutilating operation, destroying, to some extent, normal tissue, requiring a general anesthetic and only to be performed in the hands of the most skillful.

Halle<sup>6</sup> has obtained the same results, if not better results, with the following simple technic: This operation is done under local anesthesia by injection, and by application to the mucous membrane of the nose, followed by an infiltration through the membrane and into the sphenopalatine nerve on both sides. The mucous membrane on the inner wall of the inferior turbinate is freshened up by gentle scraping, but without destroying it, and the corresponding area of the septum is treated in a like manner. A vertical incision is made through the mucous membrane of the lateral nasal wall, running immediately in front of the middle and inferior turbinates, carried down to the bone and into the mucoperiosteum of the floor of the nose, in a horizontal direction, to end on the nasal septum. The mucoperiosteum of the floor of the nose is now thoroughly elevated throughout the length of the vertical incision, so that the antrum can be opened. Care must be taken to avoid splintering the bone. Finally, the lateral wall is chiseled through at its junction with the floor of the nose, the

chisel being inserted into the pocket formed by the elevation of the mucoperiosteum of the nasal floor. This procedure mobilizes the entire lateral nasal wall, which is now displaced forcibly inward and in contact with the nasal septum. The interior of the antrum is now open for inspection. The mucous membrane lining is not removed unless completely and thoroughly diseased. The antrum cavity is now packed with iodoform gauze to obtain a continuous apposition of the lateral nasal wall with the corresponding scarified area of the septum, until adhesions have taken place. The packing is not removed for five days, as there is an abnormal tendency to hemorrhage.

The postoperative treatment consists in keeping the lateral nasal wall in close and constant apposition with the septum. The packing is removed in five days, the antrum cleaned and the upper angle repacked. This is continued until the adhesions are firm and the antrum has granulated closed. This usually takes from five to six weeks.

As you can see, the operation accomplishes a great reduction in the size of the nasal chamber, augmented by the rolling up of the detached mucosa from the floor of the nose. This may ultimately lead to such a marked stenosis as to interfere with breathing.

It is better to leave the nose in this condition for a period of from five to six months, even if the patient is having difficulty in breathing. By this time all swelling will have subsided, and the adhesions may be cut, allowing the lateral nasal wall to retract to any desired width. The results of this operative procedure are brilliant.

One of us (Dr. Lux) had the privilege of working with Dr. Halle, during the twelve months of 1921. At this time Dr. Halle had operated between four and five hundred cases of atrophic rhinitis, and Dr. Lux saw the after results of three and four years' standing. These results were all universally successful, the patients being free of all disagreeable symptoms.

We began this work in America, a year and half ago, and have operated upon eighteen cases to date.

We had but one complication in the eighteen cases, that occurring in the first case. This was an infection in the lacrimal sac. This was easily corrected. In the last seventeen cases we have had no complications whatsoever.

The operation is simply and quickly done, taking not more than half an hour, and without pain or discomfort to the patient. The entire eighteen cases have been greatly improved. There is not one of these patients, at the present time, who has any appreciable crusting or odor, and all have more or less freedom of respiration.

The interior of the nose after the operation shows a remarkable change. Instead of the absence of the inferior turbinate it appears as if a new inferior turbinate has taken its place. The lateral nasal wall attaches itself to the septum. Instead of the dead gelatinous mucous membrane, it has changed to a red, velvety, moist and almost normal appearance.

There seems to be a rebirth of the glands of the lateral nasal wall, due to the increased blood supply from the septum. The odor disappears as if by magic. Many of these cases have reported back to the office during the course of treatment, stating that it has been two or three weeks since they had douched or treated their nose in any manner whatsoever. The results of the work depend greatly upon carrying out the routine of its technic in detail.

We have had the pleasure of showing this work to several men from other cities. Some of their results have been brilliant, while others are not so successful. Investigating the failures, we discovered that they were due to improper and insufficient packing of the antrum.

There has been only one objection ever raised to this work, and that was that the lateral wall would fail to stay in place. It is a relatively easy matter, if this should happen, to reformat the wall over again and reform your adhesions. This is such a simple performance that it can be done in a few minutes in the office.

#### CONCLUSIONS.

First—Atrophic rhinitis is not a hopeless condition.

Second—A tremendous amount of help can be given these patients.

Third—They can be returned to a condition so near normal that the crusting and odor will have practically disappeared.

Fourth—They will continue through life without their life being a burden.

Fifth—The Halle operation offers the most simple, the most reliable and the safest method as yet produced for giving real relief to a condition hitherto considered hopeless.

#### BIBLIOGRAPHY.

1. Sternberg, H.: *Ztschr. f. Hals, Nasen. u. Ohrenhkl.*, Berlin, February, 1923.  
Axissa: *Arch. Ital. di Otol.*, Naples, February, 1923.
2. Caldera, C.: *Laryngoscope*, January, 1920.  
Perez, Fernand: *Ann. d. mal. de l'oreille, du larynx*, Paris, June, 1922.
3. Fleischman: *Ztschr. f. Hals, Nasen u. Ohrenhkl.*, November 1st, 1922.
4. Bruzzi, Bruno: *Archiv. Ital. di Otol.*, December, 1922.  
Baumgartner, C.: *Schweiz. Med. Wochenschrift*, May, 1922.
5. Lautenschläger, A.: *Ztschr. f. Hals, Nasen. u. Ohrenhkl.*, June, 1923.
- Hofer, G.: *Ztschr. f. Hals, Nasen. u. Ohrenhkl.*, November, 1923.
6. Halle, Max: *Journal Laryngol. and Otol.*, Edinburg, Dec., 1921.

Case	Age	Sex	Duration	Crusting	Fetor	Pharyngitis	Atrophy	Operation	Results of operation:		
									8 months	9 months	1 year
1. J. W.	24	F.	14 yrs.	Cast of nose	Present	Present	Mild & inferior turbinates	Oct. 26, '22	Improved, retracted; secondary 90.	Crusting, fetor absent.	Crusting fetor absent.
2. L. S.	32	F.	24 yrs.	Large amount	Very offensive	Present	Advanced mild. & inferior turbinates	Oct. 15, '22	Mucosa succulent, no crusting or fetor.	Acute frontal sinus, right.	No crusting or fetor.
3. E. W. (negro)	19	F.	10 yrs.	Nose & pharynx	Very offensive	Present	Infer. turb. almost entire.	Oct. 17, '22	No crusting or fetor, slight discharge.	Left side retracted secondary.	No crusting or fetor.
4. E. B.	21	F.	9 yrs.	Present	Mild	None	Right middle deducted septum	Nov. 5, '22	Slight crusting, right.	No crusting.	Slight crusting, no fetor.
5. B. W.	25	F.	10 yrs.	Cast of nose	Offensive	Present	Marked	Nov. 19, '22	Slight crusting, no fetor.	No crusting, no fetor.	No crusting, no fetor.
6. S. E.	36	F.	19 yrs.	Nose, pharynx and larynx	Offensive	Marked	Advanced	Dec. 27, '22 submucous	No crusting or fetor.	No fetor or crusting, hoarse, voice hoarse.	No crusting or fetor, voice normal.
7. J. L.	28	F.	16 yrs.	Lower half nose	Marked	Mild	Inferior turbinates	Feb. 10, '23	Slight crusting, no fetor.	No crusting or fetor.	No crusting or fetor.
8. F. E.	20	F.	15 yrs.	Right side nose	Mild	Mild	Marked	Feb. 15, '23 submucous	No crusting or fetor.	No crusting or fetor.	No crusting or fetor.
9. A. B.	30	F.	25 yrs.	Cast of nose	Very offensive	Marked	Advanced	resection Mar. 14, '23	Small amount crusting.	No crusting or fetor.	No crusting or fetor.
10. F. N.	38	M.	27 yrs.	Large amount	Marked	Mild	Inferior turbinates	Mar. 22, '23	Crusting and fetor absent.	Crusting and fetor absent.	Crusting and fetor absent.
11. F. P.	18	F.	12 yrs.	Cast of nose	Very offensive	Marked	Advanced	April 13, '23	Small amount crusting.	Small amount crusting.	Small amount crusting.

Wasserman: All negative.  
Average age, 27 years. Average duration, 16 years.

CONSERVATIVE TREATMENT OF THE CHRONIC  
SUPPURATIVE EAR.\*

BY WILLIAM HENRY HASKIN, M. D.,

NEW YORK.

## I. PATHOLOGY.

The pathology of this disease owes its importance to two basal factors, the first being histologic, the second physical. The histologic factor is that the living mucous membrane of the middle ear is at the same time the periosteum of the underlying bone, and on account of this fact any long continued injury to the membrane will lead to necrosis of the underlying bone. The physical factor is that the middle ear and attic form a very irregular space which is difficult to drain in most cases. A comprehensive review of the anatomy and topography of this region is given by Dr. O. T. Freer. He shows that the attic offers poor opportunity for drainage, as it is built up in three stories or compartments. The first of these is formed by the anterior and posterior malleolar folds, with the anterior and posterior tympanic recesses or pockets created by them. The second story, the superior tympanic recess, or Prussak's space, rests upon these two pockets and its roof or *pars cupularis*. The inner attic is less subdivided, as it has only one story, but nevertheless it, too, may be a partly confined space and difficult to drain, for it is not only separated from the outer attic by the superior malleolar incudal body and often by the superior malleolar incudal fold, but many have merely apertures connecting it with the mesotympanum in cases in which a large chorda fold, stapedial fold and tensor fold exist.

Thus it is seen that while the mesotympanum forms an undivided, single, easily drained cavity of comparatively simple form, readily opened for direct drainage if the *pars tensa*

---

\*Read before the American Otological Society, June 4, 1924.

of the membrana tympani be perforated, the attic or epitympanum is subdivided into a number of compartments with easily blocked intercommunications and communications with the mesotympanum.

When inflamed, the normally thin mucous membrane folds forming the floor and partitions of the attic are capable of extreme obstructive swelling, the obstruction being aggravated by false membrane and pus clots; thus the normal outlets of the attic into the mesotympanic cavity may become wholly or partly closed, the pent up pus being dammed back into the tympanic antrum and mastoid cells. In addition, instead of having merely the tympanic membrane for its enternal wall, as is the case with the mesotympanum, the attic has an outer wall of bone, so that, in order to escape into the external auditory meatus, pus confined in the attic must find its way underneath the lower margin of this bony wall, that is, under the margo tympanicus, and it can only do that after perforating the ligamentous attic floor, finally having to burst through the swollen membrana flaccida above, or through Prussak's space before it can discharge from the ear. Therefore, epitympanic perforations, instead of being punched out holes, as they are in the pars tensa, are necessarily fistulous tracts that give poor drainage through the resistant dense tissue encountered and are situated high up above the processus brevis.

Chronic attic suppuration occurs in two types: In one, a large perforation of the pars tensa, sometimes involving all or nearly all of it, exists, the history of these cases being that in the acute suppurative stage the attic abscess, instead of perforating the membrana flaccida, has drained into the mesotympanum, which was also involved, and an extensive destruction of the pars tensa results. The mucous membrane of the promontory and the inner wall of the middle ear appears to be healthy in these cases, but are covered with a moderate or slightly foul mucopurulent secretion coming from above, the fetor remaining even after washing or cleansing the ear.

In the other type, masses of sodden, white epithelium, so called cholesteatoma, are seen to fill the middle ear in greater or less amount, in some cases nature seeming to have performed a complete radical operation.

Before analyzing the local pathology of the middle ear and mastoid we must consider some broad facts. It may be stated that practically all cases of chronic purulent otitis media, with the exception of the tubercular and the syphilitic, are due to a previous invasion through the eustachian tube. This is true, too, of the acute processes in the middle ear, but in the cases we are considering, there are certain general and local conditions that predispose to chronicity from the outset. The general predisposing conditions are: (1) The exanthemata, especially scarlet fever and measles, which often lead to extensive destruction in a short time; (2) any severe wasting diseases that lead to lowering the tone of the tissues; (3) the strumous condition, where there seems to be some element lacking in the tissues. Local causes are such as the following: (1) Insufficient drainage, as through a small perforation; (2) the irregularities referred to above, where we have a thickened mucous membrane and the retention of products, such as in the attic, etc., through adhesions; (3) the presence of small cells, easily occluded, in the antrum of the mastoid—the so-called antral cells, which undergo sclerosis, prevent drainage of the mastoid and thus keep up the middle ear discharge. Other local causes predisposing to chronic suppuration are, of course, those having their origin in the nasopharynx, occurring especially in the young, and due probably to infected lymphoid tissue. This would include diseases of dentition, catarrh, pansinusitis.

Should the primary attack or repeated acute attacks be only moderate in duration and intensity, areas of normal epithelium will soon reappear, and with the cessation of the attack will restore the cavity and thus recovery will ensue. In such cases there is probably not even a periostitis present, but simply a superficial inflammation of the mucous membrane. This is the ideal condition. If, however, the cause continues to operate with continued hyperemia, sepsis and discharge, the epithelium will undergo changes, assuming the squamous type. These changes are due to constant irritation, probably from the tryptic products of dead leucocytes and bacterial toxins, or from the extension inwards of epidermis. Should these squamous cells accumulate, a cholesteatomatous process is established, which usually, sooner or later, involves the antrum.



and eventually the mastoid, and this is usually accompanied by marked thickening of the mucoperiosteum. So long as a cholesteatoma remains free from reinfection it may cause no symptoms, although usually it is a progressive condition and becomes infected, or the pressure of the accumulating mass will cause absorption of the surrounding bone by pressure.

Reversely, should excessive and rapid desquamation cease and the scales find a ready exit, the antrotympanic cavity will retain its thin squamous lining, but owing to excessive transudation of lymph and imperfect drainage it will remain constantly moist and become a typical "septic tank" containing an albuminous fluid, a suitable media for bacterial growth. Such a condition will be dealt with under the bacteriology.

Should the inflammatory activity in the mucoperiosteum continue, a chronic exudative process is established, such a condition being characterized by increased swelling of the membrane, most marked around the fenestra, the ossicles, the opening of the eustachian tube and the aditus and antri, which eventually gives rise to a low grade granulation tissue. It is to the obliteration of the fossula rotunda and the pelvis oralis by such tissue that such marked deafness supervenes in some cases. When this is locally accentuated in the shape of pedunculated masses and polypi, we may suspect that there is a true necrosis present. The discharge in such cases is very characteristic, being marked by the presence of leucocytes, old and new polymorphonuclear and large mononuclear cells, with a high percentage of lymphocytes, whose presence is strong evidence of the existence of granulation tissue. Endothelial cells, plasmocytes and a small number of phagocytes may also be seen with all types of bacteria. If this condition persists, the bone is sure to be sooner or later implicated, because, as pointed out above, the bone is dependent on the mucoperiosteum for its nutrition. Very often again here we have a suitable media for the formation of cholesteatoma. This type is at all times liable to acute exacerbations owing to reinfection from the nasopharynx with the ordinary pyogenic organisms, especially the hemolytic streptococcus and the pneumococcus, thus explaining the mixture of organisms found in such cases. The discharge changes in character, assuming that of an acute suppuration with excess of new poly-

morphonuclear leucocytes and phagocytes, phagocytosis in such cases very often approximating to that in the acute infections. Owing to extension and intensification of the process, the surrounding bone is almost sure to be involved, especially the cancellous areas in front of and behind the labyrinth, and, of course, the mastoid.

With regard to the osseous lesions, they vary widely in situation, extent and type. Whenever granulations are persistent, bare bone is usually present. If due to caries it probably will show the bone to be rough, spongy and painful, while a firmer, smoother surface will be suggestive of death in mass or necrosis, thus approximating the condition found in acute suppuration of severe intensity. Necrotic bone also yields a greater factor in the discharge, but the cytologic changes are more marked in acute osteomyelitis in the shape of myelocytes. Caries will often be found in the ossicles, tegmen tympani, tegmen antri, sigmoid groove, antrum and the adjacent part of the mastoid, and in the cancellous bone behind the labyrinth. It may also, of course, extend to the adjacent parts and give us the usual complications.

Thus a knowledge of the nature of the discharge is of no small value, but is practically indispensable.

## II. BACTERIOLOGY.

The big question here is to know what part the various bacteria play in the production and the persistence of such a condition. Undoubtedly, the majority of cases are caused primarily by the pyogenic bacteria, the original organisms being streptococcus hemolyticus, the various groups of pneumococci, the staphylococci and the tubercle bacillus. Other factors, enumerated in the pathology above, tend to keep up the infection. The exact influence of microorganisms in the induction of chronicity is still an open question. It may be stated as follows: The acute attacks are due to the above organisms. Then other elements predispose to chronicity. The complications, such as brain abscess, sinus thrombosis, etc., are due again to the above organisms. There is one type of case, not usually recognized, due to constant reinfection of the middle ear from the nasopharynx, which gives us the "septic tank" referred to above. The discharge is very profuse, very thick and ropy,

and seemingly inexhaustible. The proper care of the nose and throat will clear these cases up almost miraculously, and yet this is the type often operated upon for radical mastoid. In such cases there is usually no true necrosis of bone, the pathologic change consisting of a thickened mucoperiosteum, due to constant irritation, and when the cause is removed the condition improves. The discharge in such a condition is highly typical, very profuse, opaque, intensely fetid, containing swarms of the various mouth organisms. There is an entire absence of leucocytes, however, and cytologically the findings consist of a few squames.

Special mention should be made of tuberculosis of the middle ear. The secretion in tubercular ears is generally very profuse and like real pus, and has the characteristic sweetish, acrid odor found in other discharges from this disease. There is no excuse in these days for not identifying the true nature of the disease if there is a laboratory that is fairly equipped within reach, for with Petroff's media, which was the original media used at the Manhattan Eye and Ear Hospital by Dr. Dwyer in our first case, and with Miller's later media, made from the spleen and lymphatic glands of animals, it is easy to get a growth in from ten to twenty days, and in many cases it is easy to find the bacilli from the secretion itself after it has been treated by antiformin and then stained.

One must always bear in mind the fact that in all long standing cases of discharge there is an actual change in the epithelial lining of both the canal and the middle ear, the squamous cell replacing all other types so that a true anatomic cure never is obtained. There is always a tendency to the formation of a dry scale throughout the canal, and it is very often a most annoying condition, causing intense itching, and when it forms over the membrana tympani, it causes a marked degree of deafness which disappears on lifting the scale away. This same condition will always be found in all radical operation wounds.

With this brief outline of the anatomy, pathology and bacteriology we will take up the conservative methods of treatment, and I will introduce here what others have been advocating in recent years with abstracts of the writers' views.

## IONIZATION.

This method has been advocated by A. R. Friel, A. G. Wells, H. L. Warwick, J. B. Kantoer, Stephen Young of Edinburgh, José Maria Barajas y Vilches of Madrid, all of whom follow the method of Dr. A. R. Friel, which is given in abstract from an article in the *Lancet*, March 3, 1923:

"Zinc Ionization in Treatment of Chronic Suppurative Otitis Media.—This treatment has an advocate in A. R. Friel. To determine which cases of chronic otorrhea are suitable for the application of this method of treatment, we have to decide on the cause of the chronicity:

"1. Is this due in any particular case to the irritation of septic fluid in the ear?

"2. Is it possible to fill the entire cavity containing the septic fluid with zinc solution, or is a portion of the cavity inaccessible without operation?

"3. Is there any factor in addition to sepsis responsible for the continuance of the suppuration, such as polypi, or granulations, or cholesteatoma?

"4. Are areas—the nasopharynx, nose and mouth—which communicate with the ear, in a septic condition? This would make reinfection of the ear almost inevitable and a continuance or a recurrence of the discharge almost unavoidable.

"Zinc ionization is an antiseptic method of treatment, and if we are to form our judgment by the results obtained in cases theoretically suitable, we are bound to admit that it is efficacious. Friel estimates that the percentage of cases of chronic otorrhea in children due to simple tympanic sepsis is approximately 50 per cent. These cases can be cured in two or three visits. In a further 25 per cent, the otorrhea is kept up by sepsis with polypi or granulations, or with mucopurulent rhinitis or inflamed tonsils. In these cases it is desirable to convert the dual or triple condition of sepsis plus polypi, or sepsis plus rhinitis, etc., into the single condition, sepsis, before attempting to sterilize the middle ear by ionization. These patients will require on an average from six to eight visits. The remaining 25 per cent are those in whom there is sepsis in a position where it is not possible to intro-

duce the zinc solution without some form of operation. These include most of the mastoid and attic cases.

"With regard to the question of enlarged tonsils and adenoids favoring the continuance of otorrhea, the writer has observed at the ionization clinics many patients in whom the tonsils and adenoids have been removed without cessation of the otorrhea, and in whom ionization readily produced a cure. Frequently other individuals are cured by ionization alone, although there is moderate enlargement of the tonsils. A small number of cases of otorrhea with mucopurulent rhinitis or inflamed tonsils are seen, and in these ionization of the ear without treatment of the nose or throat seldom effects a cure. Friel does not believe from experience that moderate enlargement of the tonsils, or a moderate degree of adenoids apart from inflammation, prevents the cure of otorrhea by ionization, or has any considerable influence in causing a recurrence of the discharge."

The writer has never had occasion to use this method, which has been used by the dental profession for years in the attempt to sterilize the root canals, but, judging from a careful study of the voluminous literature that has appeared in their journals, it has not been successful. Of late iodine has been substituted for the zinc sulphate that has been used, and this would seem to be much more rational when the value of iodine as a bactericide and also as a **great tissue builder** is remembered. Lugol's solution is used and the current is reversed from that when zinc is used, the negative pole being placed in the solution and the positive pole being on the wrist. No mention of the use of iodine in ionization of the ear has appeared, but it would seem to be well worth trying.

Aniline Dyes.—Many years ago Dr. H. Holbrook Curtis recommended methylene blue as almost a sure cure. After drying the ear with cotton he blew in a small quantity of the powder. The writer saw several of these cases after Dr. Curtis had retired, and there was no question that they had remained dry over long periods. The great objection appeared to be in the staining of all tissues, which persisted for many weeks and obliterated all landmarks.

Recently Dr. J. G. Callison has recommended a solution of mercurochrome to be used by the patient. He states that a

watery solution should not be used, and gives the following formula, which will not cause caking in the canal:

Mercurochrome-220, gm. 1; water, cc. 70; glycerin, cc. 30; alcohol, 100; misce, ft. sol. Sig., 5 drops in the ear after irrigation with boric solution, wipe out after five minutes.

He states that the stain can be removed with ordinary vinegar or a weak solution of acetic acid.

The writer has used a solution which was found to be the most perfect bactericide of many tested by the National Dental Research Laboratory and found it very satisfactory except for the inevitable stain. Berwick's Sol.: Chrystal violet, gr. V; Brilliant green, gr. V; 50% alcohol, oz. 1.

Whether bacteria have much influence in keeping up the discharge in most chronic cases is very doubtful, except in acute exacerbations, hence the use of these dyes does not seem to be rational, and it is very probable that the chief benefit is derived from the alcoholic content which dries the tissues and stimulates a return to healthy condition.

Bismuth and Iodoform.—Dr. F. Stokes, in the August 2nd Lancet, 1919, strongly advocates insufflation of equal parts of these drugs after carefully drying the canal, but the disagreeable odor and the danger of caking of masses in the ear and canal do not appeal to the writer.

Suction Treatment.—It is gratifying to the writer to note that Dr. Ery Luscher in the Berne Clinic, and W. Stuart-Low, in England, and also Dr. J. Kirkendall of Ithaca, N. Y., all recommend use of the suction pump in the treatment of all otorrheas.

In 1910 the writer presented his views on the value of suction before this Society, after using it for many years, and they have never changed. In his hands suction is the one great sheet anchor in handling all cases of purulent secretion wherever found. In the ear it is the only method by which one can really clean the canal and the tympanic cavity of secretions of all sorts without distressing the patient, if reasonable care is used, and when once this is done the application of any medication will actually reach the diseased membranes.

Yankauer Operation.—Much was hoped for when this operation was first brought forward but it has not been mentioned

for years and it probably did not prove to be the sure cure that was expected.

**Vaccines.**—In the *Travaux Scientifiques* of Dr. G. Portman for 1924, the conclusions of four articles on the value of vaccines in otology are given. They all agree that the autogenous vaccine is of greatest value in the chronic cases, whereas the stock vaccine accomplished the results in acute cases.

Recently it has been discovered that a filtrate of bouillon cultures when applied locally will cause a disappearance of all bacteria in very short order and much is expected from further investigation along this line. It is an interesting fact that these filtrates are absolutely sterile themselves and that bacteria will not grow in them.

In tuberculosis of the ear tuberculin should always be administered, beginning with infinitesimal doses and gradually increasing them, carrying on this treatment for a long period of time, with weekly intervals between doses. This treatment is of great value in the after care of mastoids in tubercular children.

In the *American Journal of Surgery*, July, 1914, the writer reported the results obtained by him on a series of cases following the lines given by Dr. Nagle of Boston, and he has continued their use in all obstinate cases with about the same results, some responding wonderfully and some with no results, in spite of which he feels that they should always be given for more than 50 per cent are undoubtedly benefited.

**Ossiculectomy.**—This operation has proven of great value in a great many cases and should always be resorted to whenever definite caries of the ossicles can be demonstrated and when they, with their bands of adhesions and ligamental folds, act as an obstruction to the proper drainage of the attic and antrum. He has always been content with the removal of the malleus and incus and has not removed any of the annulus or canal wall as advocated by some. Given a canal of reasonable size and fairly straight, these ossicles can be removed under local anesthesia with the patient in the upright position, for there is but little hemorrhage and this can easily be removed with a small catheter on a suction apparatus of any sort. The chief thing to be sure of before attempting this procedure is that your instruments are really in perfect con-

dition, with cutting edges that will cut and not tear through the tissues and cause traumatism where it should be avoided.

The results have been most satisfactory in the majority of cases both on the discharge and frequently on the hearing.

With the foregoing outline of methods of treatment, it is interesting to review the many articles which have appeared on this subject if one had time to do it.

Dr. Borden, writing in 1920 on the classification of otitis media, which appeared in the *American Journal of Surgery*, seemed to be rather pessimistic as to the cures of chronic suppurations except by the radical method.

On the other hand, Dr. Bozer, in the *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, March, 1923, reports the results of conservative treatment of 190 cases at the Mayo Clinic and gives 50 per cent cures and many others benefited. He calls especial attention to the reinfection that occurs from improper blowing of the nose which Dr. Lillie describes in his paper on Tuborrhea.

It has been a very difficult problem to determine just what to say on the subject assigned me by your president, so the preceding remarks have been gathered as showing what others are doing in these cases. Now for my personal observations covering 29 years of work, mostly at the Manhattan Eye and Ear Hospital. In 1915 I tabulated all cases seen by me up to that date and found that I had seen over 800 cases of suppurative ears, and that I had only operated upon 21 for the radical mastoid.

In all of my private practice I have never done a radical mastoid and cannot recall a single case that I have ever had cause to regret not doing so. You may realize from the above statements which you can verify if you wish to take the time, why it is that the conservative methods appeal to yours truly. There is no question whatever that very serious complications can and do arise in the course of chronic suppurations in a great many cases, but it has been my great good fortune not to have had to experience them often and never in my private work.

I think we will all agree that the removal of adenoids and tonsils when diseased is imperative in all ear diseases, and that all cases of nasal pansinusitis should be very carefully treated



because of the grave danger that exists in those cases from that condition alone, but if the patients are taught not to blow their noses too hard it is doubtful whether the pansinusitis has much influence on the cure of the suppurative ear. The ruthless slaughter of the poor turbinate, spur and deflected septum which may be causing some obstruction to breathing is utterly wrong and absolutely uncalled for in the great majority of these cases which we are considering—that is, in the opinion of the writer judging from his own personal experience in over 1000 cases. He thoroughly agrees with Dr. Shambaugh, when he states, in a paper on otitis media suppurativa chronica which appeared in the Wisconsin Medical Journal of February, 1921, that "he has never seen a case in which there was any evidence supporting the view that intranasal obstruction has an unfavorable influence on middle ear suppurative disease."

A careful study of the secretions should always be made in order to determine whether caries is present, whether tuberculosis is the cause, whether the discharge is mostly mucous in character indicating tubal and membranous disease, and to determine just where the discharge is coming from. Merely washing the canal is not sufficient, for one can never actually dry the canal and tympanum with cotton applicators. Irrigation is useful in securing the secretions for examination, but after this is done the remaining secretion should be removed with a small catheter on a suction pump, and then suction through a close fitting Siegle otoscope will reveal where the secretion is coming from. It is interesting to note that an examination of the first secretions from the canal will often give no information as to the bacterial infection, whereas the secretions drawn down by the otoscope will almost always give definite growths of pathogenic bacteria.

The X-ray is not very reliable in long standing cases of suppuration but it will reveal the presence of fairly large masses of cholesteatoma, and when the secretion is very abundant and is purulent it will show necrotic masses of bone and sometimes will reveal areas of actual destruction of the mastoid cortex. It will also give a picture of the size of the mastoid and one can generally tell whether the disease has existed from childhood, as these cases will be found sclerotic in type

and infantile in size. This conclusion has been the result of studying a long series of cases with Dr. Law, an X-ray having been taken of many of the cases in our series of vaccine cases, and it bears out Wittmark's conclusions in his studies of the pneumatization of the mastoid.

Polypi have always been removed very carefully with a snare, constriction being applied slowly and the stumps being touched with a strong silver nitrate solution to seal the raw surface. No complication has ever been encountered in this procedure although it is necessary to remove several growths at different times before the tympanum is cleared, in some cases.

When there is a great thickening of all the membranes silver nitrate solutions are often sufficient, but care must be taken not to allow the strong solutions to get too near the facial canal and cause paralysis of the nerve. Active cultures of the bacillus *Bulgaricus* placed in the canal and kept there for twenty minutes or more at a time often will give remarkable results in restoring a healthy condition of the membrane, as reported in a short article by the writer in the *ANNALS OF OTOTOLOGY, RHINOLOGY AND LARYNGOLOGY*, March, 1911.

Caries of the ossicles calls for their removal when possible, but areas of caries can often be cleared by the use of enzymol with its dilute hydrochloric acid component. This solution should also be used for periods of twenty minutes or more twice a day, the canal being filled and the patient recumbent with the ear up.

When the tube is found to be active in the production of the discharge, much can be done by irrigating through the tube from the ear with solutions of argyrol or of neosilvol. This can be done by placing the solution in the tympanum and forcing it through with pressure through the Siegel otoscope until it reaches the pharynx. These are the cases where removal of adenoids and tonsils is generally indicated, and the patient should be instructed about the danger of blowing secretions out from the nose.

As stated in the report on vaccines mentioned before, the personal element seems to play an important part in the successful care of these cases. After gaining the full confidence of the patients they should be made to fully realize the dangers

that may result from neglect on their part, and they should be advised to see their otologist at least once in three months, even though the ear appears to remain dry, for dry scabs will generally form in that time and if they are removed soon enough the recurring discharges will not occur, the discharge being due to the irritation caused by their accumulation which in time sets up inflammation in the covered tissues in an effort to throw off the irritant.

Watery solutions have been discarded for a long time by the writer except when a competent nurse is in attendance, for the one essential thing we strive for is to keep the area dry, and this will not be accomplished when these solutions are used owing to the great difficulty in drying the tympanum which we find even with a speculum in place and with perfect light.

Water causes cholesteatoma to swell and thus block the secretions in the deeper areas and this has undoubtedly frequently been the cause of very serious complications.

Alcohol will dissolve these masses and when used with equal parts of hydrogen peroxide, filling the canal and retaining for fifteen minutes at a time, much benefit generally follows. The removal of every particle of debris that can be reached is important and is facilitated greatly by a dental instrument, an excavator, that seems to have just the right angle to reach hidden corners. It is remarkable what relief is obtained after thorough removal is obtained, and in many cases by delicate manipulation a complete cast of the canal membrane can be removed even from the remains of the membrane itself and from the deep pockets above the ossicles.

In a few words, the method of treatment that has been so successful in my hands is, first, remove all secretions with suction, instruments for elevating the dry masses and then with small alligator forceps, careful sterilization with 75 per cent alcohol; drying with a current of air of low pressure and not too long application to produce vertigo and then dusting the cavity with a mixture of equal parts of nosophen and compound stearate of zinc.

This powder should be omitted at the last treatment after all secretion has ceased.

It seems hardly necessary to say that careful attention should be paid to building up the patient's general health in every way possible.

There are three principal things to be considered in the treatment of these cases, first, the danger of serious complications; second, the preservation of hearing and third, the curing up of the discharge.

Whenever there is any indication of pressure, of meningitis or of labyrinthine involvement there should be no question of the advisability of doing the radical operation.

When these indications are not present one should consider what method will give the greatest promise of preserving the hearing that is present and of even improving it in many cases, and there is no question, in your writer's mind, that local treatment with ossiculectomy when indicated will give the best result to the patient.

The cure of the discharge is the last question to be considered and this depends largely upon the thoroughness with which each treatment is given by the doctor. Many of these cases only discharge at intervals some because of a long continued accumulation of cerumen or epithelium which finally sets up an inflammation in nature's effort to throw it off, others because of a reinfection of the mucous membranes through the eustachian tubes, and method of handling these cases has already been given.

It is not at all rare to have patients come for deafness who don't even realize that they have a discharging ear until it is shown to them and they smell it when removed on an applicator, and it is often found that these cases respond most satisfactorily to treatment.

The plea that is made by the writer is that each case be given the personal care of the physician himself as frequently as necessary to clear the discharge and that the treatment should not be left to the patient or the parents to carry out until the discharge has been stopped. When this has been accomplished they can be instructed how to look after themselves and be asked to return for examination at regular intervals, in order to prevent the accumulation of hardened masses of secretion which will eventually set up inflammation again.

## THROMBOSIS OF THE LATERAL SINUS.

BY J. B. NAFTZGER, M. D.,

SIOUX CITY.

In considering lateral sinus thrombosis we will include phlebitis and infections of the sinus and internal jugular vein.

Infection of the lateral sinus with its possible complications is of sufficient frequency and of such grave importance as to justify a review of the literature on this important subject. The frequency of sinus thrombosis occurring in suppurative mastoiditis is given by Hill as 7 cases in 166; Downey, 5 cases in 79; Gerber, 25 in 524; Welty, 3 in 100, and the Massachusetts Charitable Eye and Ear Infirmary, 19 in 497. During the past four years the writer has found 6 cases of sinus thrombosis in 192 mastoid operations. Because the structure and relationship of the lateral sinus have much to do with the modes of infection and subsequent course of the disease, a brief anatomy will be considered. The lateral sinus is composed of two portions—the transverse sinus, extending from the occipital protuberance to the posterior inferior angle of the parietal bone, and the vertical portion (sigmoid sinus), extending from the posterior inferior angle of the parietal bone to the jugular bulb. The right lateral sinus is usually larger than the left. It is continuous with the superior longitudinal sinus, and is the one most frequently thrombosed. The left lateral sinus is continuous with the straight sinus and may be quite small. There is usually a small communication between the two lateral sinuses, either through a small branch or through a fenestra in their walls, where they are applied one against the other.

The lateral sinus receives the following veins: (1) Numerous small veins from the mastoid cells, (2) mastoid vein, (3) posterior parietal diploic vein, (4) superior petrosal sinus, (5) some veins from the temporosphenoidal lobe, the cerebellar hemisphere and the medulla. The superior petrosal extends from the posterior end of the cavernous sinus to the

lateral sinus. It receives veins from the region of the labyrinth. The inferior petrosal sinus extends from the posterior extremity of the cavernous sinus to the inferior jugular vein.

Poerrier gives the following description of the jugular bulb: "The inferior jugular vein begins at the foramen lacerum posterior. This is divided by bone and fibrous tissue; the posterior and larger part transmits the lateral sinus, which in going through becomes the internal jugular vein; the smaller part transmits the nerves and inferior petrosal sinus. The jugular foramen is continued into the recess, the jugular fossa, which contains the jugular bulb. The jugular bulb has venous connections with (1) the inferior petrosal, (2) the vena aqueductus cochleæ, (3) the occipital sinus, (4) vena condyloideæ posterior, connecting the bulb with the spinal plexuses, and (5) the vena condyloideæ anterior, connecting the bulb with veins within and without the skull. Thus the jugular bulb is in venous connection with the cavernous sinus, the internal ear, the venous plexuses of the cerebellar fossa and of the spinal canal and beneath the basi sphenoides. The bulb of the internal jugular vein lies below the tympanum and is sometimes in direct contact with the tympanic cavity because of a dehiscence of bone in the floor of the tympanic cavity. Thus on account of the close proximity of the sinus to the ear, infection can easily be spread to the internal jugular, to the brain and to the veins and sinuses of the vertebral column.

As pointed out by Sir William Milligan,<sup>10</sup> the lateral sinus is an interdural structure and is consequently more vulnerable than a structure separated from the focus of infection by the full thickness of the normal dura.

Modes of Infection of the Lateral Sinus.—Sinus infection is usually transmitted from the infected mastoid cells, either by the smaller veins in contact with the infection or by direct contact of the infectious material with the sinus wall. Alexander states that otitic sinus thrombosis is caused by an infection of the sinus contents by continuity or by metastasis from middle ear suppuration. By infection of a smaller vein, the infection may extend directly to the large sinuses. However, many other avenues of infection are mentioned by other observers. In cases of large extradural abscesses the sinus is compressed

and thereby mechanically a sinus thrombosis is produced. Friedman and Greenfield<sup>4</sup> cite a case of mastoiditis where at the first operation they found the sinus normal, but there was a thrombosis of the mastoid emissary vein which stopped short of the sinus. Later the lateral sinus became infected from the thrombosed mastoid emissary vein. Friesner<sup>5</sup> reports a case of sinus thrombosis following otitis, where the mastoid contained no pus and the bone over the sinus was normal. Dench<sup>6</sup> states that direct invasion of the blood stream usually occurs to the lateral sinus and that such invasion may occur directly from the middle ear through the jugular bulb without any mastoid involvement. Mosher<sup>7</sup> reports a case of thrombus of the internal jugular vein secondary to a retropharyngeal abscess, and cites Goldman's case of thrombosis of the internal jugular following an acute tonsillitis. According to Kopetsky,<sup>8</sup> in 20 per cent of the cases of thrombosis in children there is a dehiscence in the bony tympanic floor, leaving the mucous membrane of the middle ear directly in contact with the wall of the dome of the jugular bulb. Phillips states that infection takes place through (1) anatomic dehiscences of bony tissue of the parietal wall, (2) direct extension, (3) involvement of the smaller veins of the diseased bone, (4) involvement of the intermediate anastomotic veins. Boenninghaus calls attention to the fact that infection may proceed from the labyrinth directly toward the bulb by involvement of the lymph spaces of the middle ear or through a thrombus from the auditory vein.

Pathology and Bacteriology of Sinus Thrombosis.—Thrombosis is usually preceded by a periphlebitis, then a phlebitis or endophlebitis which is followed by the formation of a clot on the inner wall of the sinus. Campbell<sup>11</sup> gives the following description of the formation of the thrombus: "At first there is a phlebitis—an infection of the tissue making the wall of the vein, then comes a breach in the inner lining of the wall, a slowing of the blood current and formation of a coagulum. This coagulum becomes infected and a thrombus is formed and the way opened to a general bacteremia. The coagulum grows until a mural clot is formed, and particles of this may be detached by the blood current and thrown into the general circulation, causing symptoms of bacteremia." The whole lumen

may be occluded by a clot, the thrombus may be extensive, going down into the jugular or above toward the torcular, or both, and may also invade other communicating sinuses. It may undergo liquefaction, forming pus, or may become organized. Braun found that bacteria were present mostly in the periphery of the thrombus and that the younger thrombi contained few, if any, bacteria, thus indicating that the thrombus was formed and later became infected.

Clotting is favored, according to Hayman,<sup>12</sup> by (1) slowing of the blood stream, (2) increase of agglutinability of the blood platelets, (3) increase of coagulability of the blood plasma and by changes in the vessel wall.

After the clot is formed it may break down in the center or liquefy and the infected contents rupture through the wall of the sinus, or it may rupture through the visceral wall, forming a cerebellar abscess or meningitis. The sinus may become obliterated by an organized thrombus, or the thrombus may become partially absorbed and the circulation reestablished. In cases of direct infection from anastomosing veins the thrombus may spread directly from the infected vein into the sinuses, or particles may be detached or thrown into the blood stream.

Most observers find that the streptococcus hemolyticus is responsible for the majority of cases of thrombosis or phlebitis; the clot at first is not infected, but is soon invaded by the microorganisms. If the thrombosis is of otitic origin and the streptococcus is found in the mastoid or middle ear, the same organism can invariably be found in the thrombosed sinus. Schwartz<sup>13</sup> states that very rarely is thrombosis associated with other organisms than the hemolytic streptococcus and at times hemolytic staphylococcus. Oppenheimer<sup>14</sup> reports that in 150 cases of sinus thrombosis no other organism than the streptococcus has been found in the blood cultures, and Sir William Milligan<sup>10</sup> goes farther in stating that the causative agent in sinus thrombosis is invariably a streptococcus.

Complications and Metastasis.—Bacteremia is practically always associated with a sinus thrombosis at some stage of its formation and is so constant as to be mentioned by many as a symptom. Extension of the thrombus to the jugular bulb



and internal jugular vein and other contributory veins and sinuses frequently occurs. The jugular vein, like the sinus, may be completely thrombosed or only partially occluded, thus permitting infectious particles to be carried into the circulation, with the resulting bacteremia or metastatic abscess. Of the metastases, intracranial complications far exceed the others in number and importance, and purulent meningitis occupies the first place among these. Lung abscess, joint or muscle involvement, infarct to the kidney and splenic abscess are given in the order named. Brain abscess, with or without development of a meningitis, may develop from direct extension of the infection through the visceral wall of the sinus. Salinger<sup>15</sup> reports a case of vitreous abscess secondary to sinus thrombosis. Endocarditis is frequently observed as a complication, and nephritis is an almost constant symptom. Papillitis or choked disc is seen at times. Alexander<sup>3</sup> points out that the principal means of metastasis is by way of the jugular vein of the diseased side, but it may be through a healthy sinus by way of the jugular vein of the healthy side.

Diagnosis.—Sinus thrombosis is usually accompanied by rigors or chilly sensations and a high temperature, which is distinctly fluctuating or intermittent in type. The temperature has a tendency to show abrupt remissions and may drop to normal or subnormal, often following rather definite cycles. Profuse sweats are frequently seen. An increased leucocytosis and polymorphonuclear count is found, which is usually higher than in acute mastoiditis. Positive blood cultures are of great significance in diagnosis of lateral sinus thrombosis, but a negative culture cannot exclude thrombosis. Edema and tenderness over the mastoid emissary vein, below the tip of the mastoid and over the internal jugular, is a rather frequent symptom. Crowe's signs, pressure over a normal jugular produces dilatation of the vein over the forehead, temple, eyelids and sometimes the veins of the retina, while pressure over a thrombosed jugular gives no change, is sometimes noted. The above symptoms are given as usual typical symptoms, but atypical cases occur with such frequency that many other symptoms must be kept in mind in making a diagnosis of sinus infection. The atypical cases may have very little fever, or none at all, and little increase in the leucocyte count. In fact, they may

show practically none of the symptoms usually given as typical. Atypical cases occur more frequently in children than in adults, probably due to the extreme thinness or dehiscence in the tympanic floor which allows the infection to come in direct contact with the jugular bulb. Harris<sup>16</sup> and Tobey<sup>17</sup> divide cases of thrombophlebitis clinically into three types in relation to the occurrence of chills and temperature; (1) temperature may be high with abrupt remissions, (2) the temperature may be little elevated, chills absent, (3) no temperature. Tobey also speaks of the septic explosive type, in which the symptoms are violent from the outset and of the low grade typhoid type. He reports 11 cases out of a series of 73 in which there is no elevation of temperature during the course of the disease.

The diagnosis in all cases of sinus thrombosis has been developed to a large extent by the laboratory, which gives us valuable information in determining the presence or absence of infection in the sinuses. As in other infections, the virulence of the organism and the resistance of the patient undoubtedly play an important part in the development of a thrombosis. The value of a positive blood culture in establishing a diagnosis of thrombosis is given prominent recognition by most otologists. Libman<sup>18</sup> states that a positive streptococcus blood culture usually indicates a sinus infection, and that if the sinus has been operated on and the patient does not do well and there exists a positive blood culture, it indicates that the focus of infection has not been reached. On the other hand, we must not forget that a sinus thrombosis may exist with negative blood cultures. Kopetsky<sup>8</sup> cites a case which had repeated negative blood cultures with thrombosis of the sinus and veins. The sinus was exposed because of the marked septic symptoms and the alarming condition of the patient. He says further that where the primary focus of infection is found at the operation to reach and include the sinus, and when later unusual symptoms develop unaccounted for by lesions elsewhere in the body, the sinus should be explored. It must not be forgotten that other infections, especially endocarditis, may give positive blood cultures. Oppenheimer<sup>14</sup> and Spencer state that it should be possible to detect the bacteremia in every sinus thrombosis case at some time during the course of the disease. They report streptococcus found in 150 cases

of thrombosis. In making the diagnosis of sinus infection after excluding other body complications, Phillips<sup>9</sup> places great reliance on bacteremia, leucocytosis and a high polymorphonuclear percentage. He considers the presence of a bacteremia the most convincing symptom.

It is a well known fact that in many cases of acute mastoiditis, at operation the sinus wall is found exposed and possibly covered with pus and granulations, and no symptoms of sinus infection ever developed. On the other hand, a thrombosis may exist where the sinus wall appears perfectly healthy and the overlying bone is intact and to all appearances normal. This can often be explained by the fact, as pointed out previously, that the infection may proceed direct to the sinus from the smaller veins and the sinuses in and about the infected area. While it is true that suppurative otitis media is associated with lateral sinus and jugular thrombosis in the majority of cases, it is also true that thrombosis does occur at times where there is no evidence of middle ear or mastoid involvement. Mollison<sup>19</sup> and Pollack<sup>20</sup> report cases of sinus thrombosis where there had never been a perforation of the ear drum or discharge from the middle ear.

Therefore the question of making a diagnosis of sinus and jugular infection may be very difficult, and the history, general symptoms, laboratory examination and physical signs must be taken into account. Phillips emphasized the difficulty of a diagnosis in children with double mastoiditis and advises exposure of both sinuses and aspiration of the blood from both sinuses for examination and culture, to see which side has the excess of bacteria so that this side could be operated on if necessary. If no fluid can be obtained from the sinus, a diagnosis of thrombosis is probable. As a rule, little positive evidence as to thrombosis or the course of the infection can be gained from the condition of the mastoid or the bone covering the sinus. As Emerson<sup>18</sup> points out, dangerous types of sinus infection may not have thrombosis and, on the other hand, extensive thrombosis may exist without proving so dangerous. Pulsation of the sinus wall is not a reliable sign, as a small mural clot may exist in the sinus which does not block the blood stream.

Opening a suspicious sinus for diagnosis or aspirating blood from the sinus for examination and culture is favored by many and opposed by some. The writer has never seen bad results from either procedure. The possibility of infecting a normal sinus must not be overlooked, but in cases of double mastoiditis, with the possibility of a bilateral sinus thrombosis, aspirating fluid under careful technic seems advisable, as it is very important to know which internal jugular to ligate if the condition of the patient demands interference. The successful opening and the packing off of both sinuses has been reported in several cases. Tobey<sup>21</sup> reports a case where the sinus was opened and the jugular ligated on one side, and six days later the sinus was opened and packed off on the opposite side. Tobey states only three other cases of double sinus operation are reported in the literature since 1890. Only one jugular was ligated in three of these cases mentioned, and in one the sinuses were opened and packed without ligation. McCoy<sup>22</sup> reports opening the sinus in a case where the jugular had been ligated on the opposite side two years previous. In a study of the collateral circulation after ligation of the internal jugular, McCoy finds that the external jugular plays an important part in the return circulation. As pointed out by H. J. Prentiss, there is a tremendous variation in the arrangement of the veins communicating with the lateral sinus and internal jugular, and this variation may exist on the opposite sides of the same individual, so that in ligating, the operator cannot depend upon any conformation of the structures. He believes, however, from the examination of many specimens, that the communicating veins are always able to take care of the circulation after ligation of the internal jugular. It would seem wise, however, in the question of double thrombosis with the thought of ligating both jugulars, to ligate one and wait a sufficient time for the communicating veins to accommodate themselves to the increased circulation before ligating the other. Great care should be exercised in the dissection not to injure or ligate the external jugular.

Treatment.—Ligation of the internal jugular, with or without resection, is advised by practically all prominent

otologists as of first importance in the treatment of thrombosis or infection of the lateral sinus. By ligating the jugular the infection is, as it were, localized, and general septicemia and metastasis prevented. The question as to the time of ligating—whether before or after exposure and examination of the sinus, and whether a resection of the vein should accompany the ligation—is still open to discussion. Rott<sup>23</sup> in reviewing the literature on this subject, found that Barth, Politzer, Kerrison and Zufal advocated exposing the sinus and, if it seemed necessary to open the sinus, ligating the jugular before opening the sinus. Sheppard and Lilienthal advocated ligating before any chiseling was done. Allport, Whiting, McEwen, Loeb, Ballenger, Bezold and others advocate opening the sinus and, if free bleeding does not occur from the jugular end, then ligate the jugular. Tobey and Campbell favor exposure before ligating. C. C. Jones points out that statistics indicate the mortality is as great where there is a routine ligation as where ligation is reserved for the severe cases. It is agreed by practically all operators that if there are positive signs of thrombosis, a ligation should be done. In the absence of the so-called typical symptoms, if the patient is not doing well following a mastoid operation with temperature, and if other diseases have been ruled out, ligation should be considered. Ligation should be done if there is a bacteremia following suppurative otitis or mastoiditis. H. L. Baum<sup>24</sup> reports cases of opening the sinus and draining an abscess within the sinus without disturbing the thrombus. He stresses the fact that where there is no reason to suspect that general infection is developing, simple drainage, if pus has formed, or leaving the clot undisturbed is good surgery. We know that many cases have been found accidentally where the sinus had been obliterated by an organized thrombus, which had formed at some previous infection, and the patient had made a good recovery. Practically all otologists agree that ligation is, as a rule, as satisfactory as resection. A resection can be done later if needed. Dr. Dench emphasized putting the ligature above the facial veins, to cut off the possibility of a systemic infection. Phillips, in 1922, stated that he had not done any resections in five years, except in one or two

cases. In a very few cases reported, after ligation of the jugular and the thrombus had been removed from the sinus, local infection developed in the region of the bulb or in the neck, and the neck wound had to be reopened or the vein resected.

In every case after a mastoid operation, where the symptoms lead you to suspect a sinus thrombosis, a thorough and wide exposure of the sinus should be made in order to carefully inspect the condition of the sinus wall and to detect any evidence of a perisinusitis or extension of the infection to the dura.

Blood transfusions have begun to occupy an important position in the treatment of septic sinus thrombosis. Hayes<sup>25</sup> makes a statement that cases with definite symptoms of sinus thrombosis can get well if blood transfusions are given in time, and also that patients with intermittent temperature following mastoid operations without sinus involvement make good recovery following blood transfusions. He cites cases of recovery without sinus operation following transfusions of whole blood by the Unger method. Kopetsky<sup>8</sup> favors blood transfusions in severe cases with positive streptococcus blood cultures, and where bacteremia is increasing. Friesner<sup>4</sup> has also used blood transfusions with good results. Unger immunizes the donor and gives whole blood transfusions, but points out that autogenous immunization is to be preferred if time permits. McCoy (*Laryngoscope*, April, 1922) reports a case where a sinus operation was followed by slow and unsatisfactory recovery, in which rapid improvement was noted after blood transfusion. Blood transfusions have a marked influence in combating septicemia and are also of great supportive value in extremely septic cases.

The writer feels that it should not be construed that blood transfusion should supplant ligation of the internal jugular in thrombosis of the lateral sinus, but that it is a valuable adjunct in the treatment. From the reports of a number, notably Hayes, Phillips, Kopetsky and Friesner, good results have followed transfusions of whole blood in mastoiditis, with or without involvement of the lateral sinus or jugular, when the patient is septic and has little resistance.

If there is a bacteremia, transfusions may be used before or after operation on the sinus and jugular. Transfusions of whole blood by the Unger method are advised.

The general treatment of these cases must not be overlooked, as nephritis is usually present and endocarditis frequently develops. A competent internist should direct the general care in every case of sinus thrombosis.

#### SUMMARY.

1. The occurrence of the atypical cases should be kept in mind, especially in children where the anatomic position of the bulb favors infection direct from the tympanum.
2. In suppurative otitis with an unusual course and evidence of sepsis, lateral sinus infection should be suspected and a blood culture made. If the blood culture is positive, the sinus should be exposed and the internal jugular ligated to cut off the infection from the general circulation.
3. In any patient showing grave sepsis and bacteremia, other body complications being excluded, a thrombosis of the lateral sinus should be suspected; a negative blood culture in these cases, however, does not exclude sinus thrombosis, and the sinus should be inspected if other symptoms of thrombosis are present.
4. Exposure of the lateral sinus before ligating the jugular seems good judgment, and if free bleeding does not occur from the jugular end a ligation of the internal jugular should be made.
5. Ligation is preferable to resection of the jugular because it can be done in much less time and with a smaller resulting scar.
6. Transfusions of the whole blood are indicated in any case where the bacteremia is increasing or where the case is not making satisfactory progress, but is not meant to exclude ligation of the jugular or other treatment. Transfusions are especially indicated in cases of bilateral thrombosis where one hesitates to operate. Transfusion may be used before or after operation on the mastoid or sinus in severe cases to combat septicemia and as a supportive measure.

## REFERENCES.

1. Ballance.
2. Hill, F. P.: *Annals Otol. and Laryn.*, 1920.
3. Alexander, Gustav: Vienna, 1907.
4. Friedman and Greenfield: Primary Thrombosis of the Mastoid Emissary Vein with Secondary Involvement of the Lateral Sinus. *The Laryngoscope*, May, 1923.
5. Friesner, Isadora: Case of Sinus Phlebitis with Normal Middle Ear at Time of Operation. *The Laryngoscope*, August, 1922.
6. Dench, E. B.: The Treatment of Severe Septic Infections of Otitic Origin. *The Laryngoscope*, February, 1923.
7. Mosher, H. P.: Deep Cervical Abscess and Thrombosis of the Internal Jugular. *The Laryngoscope*, 1920.
8. Kopetzky: Report of Unusual Cases of Septic Sinus Thrombosis in Children. *The Laryngoscope*, 1920.
9. Phillips, Wendell C.: Diagnosis and Treatment of Septic Sinus Thrombosis. *Jour. A. M. A.*, August, 1923.
10. Milligan, Sir William: Diagnosis of Septic Sinus Thrombosis. *Brit. Med. Jour.*, 1922.
11. Campbell, Don M.: Lateral Sinus Thrombosis. *Jour. Mich. Med. Soc.*, 1922.
12. Haymann: *Archiv. für Ohrenheilkunde*, Vol. 83.
13. Swartz, Alfred A.: Contribution to Etiology of Septic Sinus Thrombosis. *The Laryngoscope*, September, 1923.
14. Oppenheimer, Seymour and Spencer, Harry: Value of Laboratory Examinations in the Prognosis and Diagnosis in Otolaryngology. *The Laryngoscope*, March, 1920.
15. Salinger, Samuel: Report of a Case of Vitreous Abscess Following Sinus Thrombosis. *Annals of Otol., Rhin. and Laryng.*, September, 1923.
16. Harris, Thomas J.: Aural Infections as a Cause of Severe Systemic Infections. *The Laryngoscope*, September, 1922.
17. Tobey, G. L.: *Trans. Am. Otol. Soc.*, IV, 1919.
18. Emerson, Francis P.: Clinical Manifestations of Infection of the Lateral Sinus. *Jour. A. M. A.*, August, 1920.
19. Mollison, William: *Roy. Soc. Med.*, London, 1920.
20. Pollock, H.: *Annals Otol., Rhin. and Laryngol.*, 1922-23.
21. Tobey, G. L.: *Am. Laryngol., Rhin. and Otol. Soc.*, August, 1923.
22. McCoy, John: Observation on Lateral Sinus Thrombosis with Report of a Case. *The Laryngoscope*, April, 1922.
23. Rott, O. M.: Concerning the Question of Jugular Ligation in Sinus Thrombosis. *Annals Otol., Rhin. and Laryn.*, 1920.
24. Baum, H. L.: Thrombosis of the Lateral Sinus Without Sepsis. *Annals Otol., Rhin. and Laryn.*, 1920.
25. Hays, Harold: The Treatment of Sinus Thrombosis With Special Reference to Blood Transfusions. *Eye, Ear, Nose and Throat Monthly*, October, 1923.



LXXVII.

CILIATED EPITHELIUM AND OTHER PROTECTIVE  
AGENCIES.\*

BY HENRY L. SWAIN, M. D.,

NEW HAVEN.

Man, the noblest of God's creatures, is—disguise it as we will—but an incident, as it were, in the life history of the streptococcus. A shrewd observer once said that nine out of every ten of us died from the immediate or more remote results of streptococcus activity, died, that they might live, and the tenth was killed by accident. (In these days of automobiles this last is too small a percentage.)

Not so all of God's creatures. To go well down the scale, alligators and crocodiles, if we may believe what we are told by those who keep them, in their primitive state were not affected by disease, and in all the thousands of years since they have been living, as they do even now, for hundreds of years, they would have overrun the earth had it not been that the way they seem to die is from injury, incurred usually in the fights among themselves, presumably over some choice bit of food or sunlight, or some very coy and flirtatious female. Except from his lusts and passions he is protected from both within and without. Reversing the human order, one-tenth of his deaths may be due to disease, and he has both teeth and tonsils, such as the latter are.

And yet we do not all die, even in these effete days. Some of us live on, even though surrounded by the unseen hosts of our adversaries, the germs.

None run amuck of more pathogenic germs than those making up this distinguished group of gentlemen, whom I am privileged to address, and who in their daily walk in life are more often coughed upon—so to speak—than most any other calling.

\*Remarks made in presenting pictures upon the screen (by invitation) before the Eastern Section of the American Laryngological, Rhinological and Otological Association, at its meeting in New Haven, January 7, 1924.

Something protects us. And if it always worked efficiently we would always remain protected and the streptococcus would not be gloating over his victims as he does now every winter. Even in the summer seasons and climes, he still may make himself notorious, and I wonder if we might not put up a better fight, if we could aid, abet and preserve protecting agencies by at least conserving them in every way. Also it may not be beneath us to stop, as I have intended to ask you to do today, to consider some of the means which ward germs from us—not with the slightest notion that I may by any chance add to the knowledge of any one of you, but that by the renewed contemplation of the subject we all may be led to stress the importance of this same conservation so that those whom we teach may be sure to get the same enlightened viewpoint which you yourselves now have.

In this eminently surgical age the operations which we do being spectacular, objective, make much more impression than anything we usually say concerning the failures of nature to protect, often because she is handicapped by the way we live and do. We ought to impress the fact that it is this failure which gives the germs the chance to not only make sufficient disease to create the necessity for operation but which also may so overwhelm us that we die.

So let us contemplate today for just a few minutes how we are protected, and what we may do to keep the system in operation, and why.

Reduced to its simplest terms, our protection against infection from without—specifically, germ invasion—is absolutely dependent upon the intact outer surface covering of the body and the mucous membrane. In the case of the skin and the more exposed areas, a heavy tough layer of cells called epidermis does the work most effectively and only when actually abraded do germs get under it.

A flat pavement epithelium covers the surfaces within the orifices of the body, and a little farther in, as in the nose beyond the vestibule, a columnar epithelium, very delicate in structure, rears itself to be, a bit further in, converted to ciliated epithelium. As this outer layer gets thinner it is constantly moist until in the nose the surfaces further in become wet. In this layer of fluid the cilia have more or less definite

motion, constantly in action, to move the fluid in the desired direction. Substances in suspension or solution in the fluid are thus not allowed to stay long in any one place—a most desirable and useful function on the part of the cilia, but you understand it is not the passive resistance which a pavement epithelium, or still more, the epidermis, presents. A layer of collodion does almost as well, if not better, but in the case of the moist membrane the protection depends on the life of the cell or rather its functional activity. And this last is no mean work. We have grown to accept these functions, so vital to our health and existence, without often contemplating the wonder of it all. Healthy membranes covered by healthy cells in a healthy body, well fed and well kept, seldom fall prey to infection. The reverse is often equally true. The equilibrium between health and disease is sometimes difficult to analyze, but, expressed in its simplest form, involves, in the case of the nose, first and foremost, the complete integrity of the outer layer of the mucous membrane. The whole protective mechanism in the nose is really one of construction and maintenance. If we erect a wooden building we can do it along the lines which make for tight joints with no place for moisture to enter and there to be retained. Such construction would tend to conserve the integrity of the structure. The whole could then be covered by a coating impermeable to moisture.

If rightly constructed and adequately protected by good paint, it would stand a long time. Deprived of its protective covering, the wood rots, the structure leans, then totters, and finally falls. So with man's nose, if the ciliated epithelium is intact, germs and other deleterious matter are wafted downwards and backwards into the throat and there disposed of. A free flow of bland serum oozes between the cells of the epithelium, and thus bathes the surfaces, helping by mere mechanical action to keep the membrane clean. In years past it has been proved that this serum in its healthy state, while not antiseptic or deadly to germs, tends to inhibit their growth. The presence of this fluid is also essential, and vitally essential to the health and activity of the surface cells and the mobility of the cilia of the epithelium. In its interior the normally constructed nose is so made that everything can be moved along and there are no retention spots. Normally there is not

much real mucus present on the surface, and this is of the thinnest and blandest type. When the danger signals are set and the invader is to be kept away or an irritant kept off the membrane, then the activity of the mucous glands gives abundant mucus to act as a coating to protect the cells and to enmesh and thus render innocuous the invaders. Matter which enters the nose may be inert, as various kinds of dust. In these days, living in the cities as most of us do, our cells become accustomed to dust. It calls forth no special reaction on the part of the cells, except that it be removed.

When in too great quantities for our very tolerant cells to endure, it is often washed away by a call being sent out to the central nervous system to supply more serum or more mucus. This call may be general or local, and may come from the epithelial cells themselves, or if they die early in the game, from the cells of the tissue beneath or surrounding the area which is out of commission.

Reduced to its lowest terms, we are, as said before, all dependent upon the integrity and in a way on the intelligence or alertness of the epithelial cells. This is particularly so in the tonsils or lymphoid areas, as has been so ably demonstrated and studied by Jonathan Wright. The question has often been asked by those who carry their investigations thus far, how can it be true that, as he has shown in the case of the tonsils, when the equilibrium is maintained, inert matter like cinabar is allowed to pass freely into the tonsils in the current of inflow without let or hindrance. Leptothritic threads are allowed to lie upon or among the cells of the outer layer, but are not permitted to pass beyond, while the pathogenic germ is not only not allowed to enter, but is actually kept off the membrane, repelled, never being found in the spaces between the epithelial cells, but outside in the crypt mingled with the leucocytes or lymphocytes which have emigrated or been washed by the outward flow of lymph. The epithelial cells themselves seem to have an absolute antagonism for these germs. Apparently only when the equilibrium is disturbed can the germs be found in the substance of the tonsil. This is quite as probably true of the active ciliated epithelium of the nose.

Now, such an action on the part of the cells seems to involve almost an intelligence, and seems beyond credence, and to explain it all sorts of theories have been most abundantly discussed. Up to the present we have known in a general way that monocellular organisms actually did what we have insisted, the epithelial cell does more or less efficiently everywhere that such potentialities are shown, as evidenced in the tonsil and the waving cilia of the epithelium of the nose, but the matter had never been consistently studied. There exists a monocellular organism to which the name *stentor caeruleus* has been given, and upon the surface of this monocellular organism exist cilia similar to those which characterize the ciliated epithelium of our bodies. A. A. Schaeffer has studied the habits of this cell under the microscope and has been able to discover substances that were pleasing or acceptable to it and also those that were not.

Apparently when so disposed the *stentor* is able to have its cilia waft towards its pouch substances which it likes or chooses, these being sometimes incorporated in the interior of the cell and later either modified, assimilated or thrown out again; but when anything comes along which the *stentor* does not choose to take in or does not like, then the cilia waft this material away from the cell body. Such things are rejected. He is able to distinguish three kinds of matter; inert, or matter to which it is indifferent, matter which it dislikes, and that kind of matter which he prefers for food.

When hungry, like his multicellular neighbors of the genus *homo sapiens*, he will eat anything it is easy to get, but when sated or when the kind of food he likes is present, he will select it and eschew the others. Thus we have the whole phenomenon of cell existence exemplified and the whole process of our protective organism typified. What this cell does each and all of our protective cells must and do accomplish for us, and on these kind offices hang our health, our very existence. Now, if we understood just how the *stentor* does this, being as he is but a single cell, how he does this great work of selection and sends his cilia vibrating one way or the other, we would be in a much better position to understand and combat disease. Wright has carried his studies and his hypotheses much further than most of us can follow him, and whether

this power of the cell is an ultimate result of mere surface tension or of the attraction or repulsion of electrically alike or different particles, or even refined chemical or chemotactic process, we must not now stop to discuss. Certainly in the stentor it seems more than a mere physical phenomenon.

When we venture a little farther afield and consider the filterable viruses of epidemic diseases like anterior poliomyelitis, spotted fever, perhaps "flu" and the latest of the group—the very frequent "common cold," we can only hope for ultra activity of the cilia so that no absorption has time to take place as again our best protection.

In presenting pictures taken of the diffuse lymphoid tissue of the pharynx and of the various tonsils, attention is called to the isolated follicles of the posterior pharyngeal wall and how each of these is potentially, both for good and evil, of the same nature as are the tonsils themselves.

Mention is also made, in showing this section of a tonsil filled with carbon particles, of how these were injected into one of the lymph nodes of the neck one hour before the tonsillectomy, and had found their way into the substance of the tonsil in this brief time. This slide was one sent by Dr. Sanford Blum of San Francisco, who has done a notable piece of work in that line and believes that what has here occurred could take place under certain pathologic conditions. This gives an added importance to the tonsils themselves and arouses one's imagination to such an extent that Dr. Winkler, under Prof. Winternitz's splendid supervision, is trying out anew the whole question. I had hoped to show more of the results today, but the work is not far enough advanced.

As the picture shows the stentor, so we may see how little he really differs from the various other ciliated epithelial cells which are present in every kind of creature, from the lazy salamander up to us of today. The ciliated cells are always where they are necessary to keep things moving along. In the nose they move downwards and backwards to the pharynx, eustachian tube, also downward, and in the trachea they work upward.

I wonder how many of you have not watched the wonderfully efficient work which they do there, and much more so in the remote tubes of the lungs. Time and again I have seen

a bunch of mucus—many times made plainer by dust particles or blood—come all the way from the carina into the larynx where a simple clearing of the throat, as we call it, sends the mass into the esophagus, or a slight cough allows it to be expectorated. These masses sometimes stick to the vocal cords where there is no ciliated epithelium, and where, as in deep slumber or prolonged speaking, they would become firmly attached were it not for the kindly ministrations of the glands with which the ventricles are freely supplied, and particularly the interior of that capacious pocket known as the sacculus.

Here, again, ciliated epithelium comes to the fore to cause secretions to be moved along to the cord and prevent the surface from becoming dry. Retained secretion, moist matter which long remains on one spot in the air passage, will inevitably become septic—usually germ laden—and always offensive and putrid if delayed long enough. In the nose this is particularly evident in the ozenas, and one of the first results of the altered secretions which come with this disease, whether like Perez and Horn, we lay stress on Perez's bacillus as a cause or not, there appears a desquamation of large areas, we might almost say, and on these areas unctuous masses of putrid matter remain until they fall away from their own weight because the cilia are not there to do duty. The construction of the nose is such that when normal the cavity would macroscopically, so to speak, drain itself readily. Enough activity of the serous and mucous glands would wash away everything. Microscopically the cilia would attend to the rest. If they are active and functioning, no need to fear infection. How different if some sudden chill or maybe the disaster of a ruthless clinician renders large sections inactive. It is surely good sense to conserve every ciliated cell. Particularly, when we contemplate the destruction and ruin which follows when a blast of cruel war gas, or fire, or fumes or grippe denudes the trachea, and way down into the tubes we have nothing left to protect us from infection, especially by way of the lymphatics of the tracheal wall.

Even the cough which racks excruciatingly in these cases can naught avail to keep the passages clear, because the secretions adhere to the areas lacking cilia.

How surprisingly alike the pathologic changes from gas and flu are, can be seen in the slides which follow, and was first called to our attention by the work done in our laboratory by Professors Winternitz and Smith. We all had known of this loss of epithelium in certain cases of grippe ever since the first pandemic of 1889-90, and each outbreak since has brought illustrative cases, but their observations do most beautifully demonstrate the important likeness of the gas and flu in the disastrous work of robbing these surfaces of their great protection—the ciliated epithelium.

Dr. Smith will lead you along with the all-seeing eye of the pathologist and will thus, I hope, serve the better to impress upon us how safe we are if our protectors are on guard, intact and actively functioning, and how much we, for ourselves and in our work for others, should always bear in mind how delicate these efficient cells are and make our therapy and our bronchial surgery effectively conserve them in their wonderfully beneficent purpose.

195 CHURCH STREET.



## LXXVIII.

### A STUDY OF THE EFFECT OF ROENTGEN RAY THERAPY UPON IMPAIRED HEARING.

BY D. C. JARVIS, M. D.,

BARRE, VERMONT.

#### INTRODUCTION.

A number of years ago, while doing postgraduate work, I was impressed by the assistance given by the Roentgen ray in a study of sinus conditions. On my return home, realizing that the nearest roentgenologist was forty-eight miles away, I purchased a Roentgen ray transformer for office use, hoping by means of its assistance to gain a better insight into the clinical conditions appearing in the course of the day's work. I happen to practice medicine in a city in which between two and three thousand granite cutters reside; in fact, the granite manufacturers will tell you that this city represents the largest granite cutting center in the world. For years we have faced a clinical problem resulting from granite dust inhalation, and, after working upon this problem for a number of years, the National Tuberculosis Association, through its Committee on Mortality from Tuberculosis in the Dusty Trades, entered the field and announced through its representative that arrangements had been completed for an investigation of the granite cutting industry as represented by the granite cutting plants of Barre, Vermont. I offered my services to this committee, stating that I would be very glad to do the Roentgen ray work or other work that a medical man situated locally might do. This offer resulted in my having local charge of the medical phase of the committee's work during the life of the investigation. As you will readily appreciate, an investigation of this kind accumulates a mass of data. There is the statistician's report, covering some 175 pages, printed by the United States Labor Bureau, the report of the United States Bureau of Mines, that of Dr. L. U. Gardner, of Saranac Laboratory, on the experimental pathology conducted upon guinea pigs, and

the report of Dr. Edward R. Baldwin, chairman of the committee, to the directors of the National Tuberculosis Association. In addition, there are a number of articles by myself dealing mostly with the Roentgen ray phase of the work. In accumulating a mass of data of this kind, one also accumulates a number of problems requiring solution, and it was while working upon the solution of some of these problems that the subject matter presented in this paper came to light.

#### FUNDAMENTALS.

In undertaking research work of a medical nature, it seems advisable to formulate fundamentals as a basis for bringing to light and solving problems. Without fundamentals it is so easy to leave the main highway of work and start detours which require sometimes almost a lifetime of work to complete. Up in the country one lives close to nature, and it is most natural that her fundamentals be adopted when undertaking medical research. These fundamentals we know are ages old and have stood the test of time. If any you reading this, follow gardening as a hobby, you realize that any flower, whether grown successfully on vine, plant or bush, requires for its successful growth the observance of certain fundamentals, which one may enumerate as follows: There must be suitable soil, there must be suitable preparation of the soil, there must be seed of sufficient germinating power or root of sufficient strength, a certain amount of moisture, a certain amount of heat and the soil at rest after preparation and planting. A study of the clinical condition occurring in the chests of granite cutters, following long continued inhalation of granite dust, discloses the fact that in order for the clinical condition to develop there must be present in the respiratory tract of these men tissue representing suitable soil, there must be suitable preparation of the soil, and, if pulmonary tuberculosis is to be superimposed upon the granite pneumoconiosis there must be tubercle bacilli present of sufficient virulence, a certain amount of moisture, a certain amount of heat and the soil at rest after its preparation. As one observes the laboratory worker, who deals with microorganisms, one notices these fundamentals of nature, for he selects suitable culture media, this media must be suitably prepared, the microorganism being dealt with must

have sufficient virulence, he takes care that there is sufficient moisture and heat, and that the soil is at rest after preparation and inoculation. After working and thinking along these lines one finds when he returns to his patients that he realizes that disease does not always appear the same in different individuals because patients represent a difference in soil, there is a difference in the preparation of the soil, the virulence of the microorganism, the amount of moisture, the amount of heat in the part affected and the soil at rest. As flowers in one garden differ in excellence from flowers of the same name in other gardens because of a difference in the degree to which nature's fundamentals have been observed, so it would seem that disease occurs or does not occur, and if it occurs is present in severity in proportion as these fundamentals of nature are observed in the individual.

#### INTEREST IN LYMPHOID TISSUE.

While working on some of the problems surrounding suitable soil, it was discovered that an individual was susceptible to the ill effects of granite dust inhalation in proportion as lymphoid tissue was present in the upper respiratory tract. That is, a young man, twenty years of age, entering the industry and disclosing on examination small sized turbinates, a smooth posterior pharyngeal wall, tonsils not large in size and no enlarged lymph nodes externally, did well in the industry; on the other hand, a young man, the same age, entering the industry and disclosing on examination hypertrophic rhinitis, enlarged lymphoid nodules on the posterior pharyngeal wall, tonsils larger in size than one would expect to find, and perhaps an enlarged lymph nodes externally, did badly in the industry and suffered early the ill effects of granite dust inhalation. In an effort to discover why some individuals had more lymphoid tissue in the upper respiratory tract than others, a great deal of work was done, and while analyzing the diet of 500 granite cutters it was discovered that lymphoid tissue was present in the upper respiratory tract in proportion as fats were absent from the diet. In checking this observation up from another angle, men who were clinically ill following granite dust inhalation were given olive oil in varying amounts and fats generally added to their diet, with the result

that one was able to observe a subsidence of cough and expectoration and a gain in weight in early cases without additional treatment. As a further check upon this observation, for the past two years the upper respiratory tract of about a hundred students in a college preparatory school has been examined and the amount of lymphoid tissue present checked up with the diet, with no change in the conclusion following an analysis of the diet of 500 granite cutters.

#### OBSERVATIONS.

While working on some of the problems surrounding suitable soil, an invitation came from the American Roentgen Ray Society to present a paper on a Roentgen ray study of granite dust inhalation at the annual meeting of the society held in Minneapolis. We had taken stereoscopically the chests of several hundred men, and a great deal of time had been devoted to a study of the films, not only in an effort to interpret the densities seen, but also to discover if possible exactly what was going on in a granite cutter's chest. While attending this meeting more or less was heard with reference to the effect of Roentgen ray therapy upon lymphoid tissue of the upper respiratory tract and, being interested in any agent influencing lymphoid tissue, I copied down the technic and on my return home started a set of observations in an effort to discover if possible how much one really could influence lymphoid tissue of the upper respiratory tract by means of Roentgen ray therapy. As a part of these observations, during the summer of 1921, a patient about forty years of age was treated by means of the Roentgen ray for an enlarged lingual tonsil, the technic used being that recommended by Witherbee, namely, a 7 inch gap, 5 ma., 10 inch distance, with the exception that a two minute exposure time was used. One week later the patient returned to the office and volunteered the statement that the right ear, which had been stuffy for ten years, was now clear and that hearing was improved. No particular importance was attached to this statement at the time because interest centered around the effect of Roentgen ray therapy on lymphoid tissue, but one month later, having a patient who at varying intervals had appeared at the office for inflations of both ears by means of the eustachian catheter

and not succeeding readily in restoring hearing by means of the usual inflations, a trial of the Roentgen ray was suggested, the area exposed for treatment being that recommended by Witherbee in the treatment of tonsils. Two days later the patient, who was the proprietor of a general store in an adjoining town, returned to the office and stated his hearing had been restored, his ears were now clear and he experienced no difficulty in hearing while waiting on customers. From this statement, interest was aroused in this subject.

TYPE OF PATIENT IN WHOM ROENTGEN RAY THERAPY PRODUCED  
SUBJECTIVE RESULTS.

As cases were treated, it became apparent that all patients complaining of impaired hearing were not benefited by use of the Roentgen ray. It was soon realized that this form of treatment was applicable only to certain types of cases, and an effort was made to discover the type of case in which the use of the Roentgen ray might be expected to bring about improvement. As one thinks over the patients complaining of impaired hearing coming to one's office, they seem, in Northern New England at least, to fall into three groups. The first group seems to include children and young adults in whom impaired hearing is so often times explained by the presence in the nasopharynx of a mass of adenoid tissue. The second group seems to include adults between the ages of twenty and fifty, who are subject to frequent head colds, complain more or less of a constant catarrhal discharge from the throat, and at times a stuffiness in the ears with the accompanying impairment of hearing. Patients of this group seem to appear at one's office at varying intervals for inflations of the ears, and oftentimes emphasize the annoyance occasioned by the presence of head noises. The third group seems to include patients advanced in years, in whom impaired hearing represents the outstanding symptom, less emphasis being placed upon throat symptoms. As one thinks over these three groups, the outstanding feature of the first is impaired nasal respiration; of the second, the prominent throat element of the case, and of the third, simply impaired hearing. As observations were continued, it became apparent that the type of patient represented by group two was the type in which one might

expect results from the use of the Roentgen ray. As patients of this group were studied more at length, it was observed that many times the turbinates were increased in size, that there were present lymphoid nodules on the posterior pharyngeal wall, and oftentimes a prominent band of lymphoid tissue running up either side of the pharynx just posterior to the posterior tonsillar pillar. In describing the type of patient in which one may expect results from the use of the Roentgen ray, you will notice a condition of the upper respiratory tract is described as an indication for treatment rather than aural condition. It seems conceivable that certain parts of the United States may have more individuals of this type present among the inhabitants than others. This may be the case in Northern New England. If observations such as these are possible, the question naturally arises in one's mind as to what is it that makes such observations possible. What underlying fundamental truth are we dealing with, when we influence certain types of impaired hearing by means of the Roentgen ray? Do we influence suitable soil by means of Roentgen ray therapy? Do we influence suitable preparation of the soil or do we destroy existing microorganisms?

#### SUITABLE SOIL.

If one cares to consider whether we influence suitable soil, then one has to decide whether we influence lymphoid tissue, because lymphoid tissue seems to represent suitable soil. At once there comes to mind all the experience of the past, with reference to the effect of Roentgen ray therapy upon enlarged cervical lymph nodes. In times past, we thought we influenced the enlarged cervical lymph node directly by means of the Roentgen ray, but with the passing of time and the focusing of attention more and more on the tonsil, it has been thought that we influenced the tonsil primarily and the enlarged cervical lymph node secondarily. Krause, in the lungs, has shown that the behavior of a lymph node is dependent upon the behavior of the lesion external to it. But one wonders if we have not jumped at conclusions when we conclude that we influence lymphoid tissue primarily by means of Roentgen ray therapy. Lederer, reporting from Beck's clinic, seems to show no marked reduction in size of tonsils occurs follow-

ing the use of the Roentgen rays, and Babcock, in a later report, in which he includes some of Coakley's cases, seems to show conclusively, by means of pathologic studies, that an abundance of lymphoid tissue exists in tonsils treated by the Roentgen ray. My own observations undertaken as a part of the investigation, rather than with any thought of supplanting tonsillectomy with Roentgen ray therapy, seem to lead one to expect very little reduction in size of tonsils from the use of the Roentgen ray. In the series of cases observed, in only one case, and then in only one tonsil, did it seem possible to enter upon the record that a reduction in size had taken place. It would seem then, from a study of the evidence at hand, that primarily we do not influence suitable soil by means of Roentgen ray therapy.

#### DESTRUCTION OF EXISTING MICROORGANISMS.

If one cares to consider whether we influence this fundamental, then there are a number of pieces of evidence deserving consideration. Last fall, while spending ten days with Dr. Kennon Dunham, of Cincinnati, looking over his pathologic and roentgenologic work on diseases of the chest, the subject of Roentgen ray therapy came up during the course of conversation, and he called my attention to an article of his, printed in the May, 1916, number of the American Journal of Roentgenology, on the treatment of carbuncle by means of the Roentgen rays. It seems possible to influence the course of a carbuncle so that the infected area soon ceases to be painful and the infected part can be moved without distress. Sometimes the induration is gradually absorbed, with little or no discharge. The same treatment is applicable to palmar abscess of the streptococcic type, and it would seem results are not from any influence of the Roentgen ray upon lymphoid tissue, but rather from a destruction of microorganisms. The work of Dr. Preston M. Hickey of Detroit on the treatment of diphtheria carriers, by means of the Roentgen ray, also deserves consideration. His work may be summarized as follows: In untreated cases of diphtheria carriers there were 34 per cent of spontaneous cures, cases treated with mercuriochrome, 68 per cent of cures; pharyngeal cases treated by the roentgen ray, 80 per cent of cures. It would seem possible

to conclude from Dr. Hickey's work that there is a diminution in the bacterial content of the throat following the use of the Roentgen ray, but if one endeavors, outside of the body, to influence directly by means of the Roentgen ray bacteria growing in colonies on ordinary culture media, we are told results are nil. A study of the evidence at hand tends to cause one to conclude that, while in the throat destruction of microorganisms is shown by bacteriologic studies to take place, this destruction is probably not the result primarily of the action of the Roentgen ray.

#### SUITABLE PREPARATION OF THE SOIL.

There remains for consideration the above fundamental, and it seems as if this is the fundamental with which we are dealing when we influence certain forms of impaired hearing by means of Roentgen ray therapy. Some time ago, while treating a small growth upon the face of a girl with very rosy cheeks, it was discovered after the treatment that there was a blanched area on the face corresponding to the area treated and approaching in size the small opening in the treatment cone. When asked how long this area remained whiter than the surrounding surface the patient replied that the condition existed for two to three days. Following this observation, the mucous membrane of the throat was studied before and after treatment, and it was noticed that it developed a grayish appearance following Roentgen ray therapy. It was also recalled to mind that one of the results to be expected from treatment of uterine fibroma was cessation of menstrual flow. If one looks up the histologic effect of Roentgen rays, textbooks on roentgenotherapy tell us that by means of the Roentgen ray we produce an edema of the endothelial lining of the blood vessels. This edema produces in the smaller capillaries a narrowing of their lumen, thus interfering with the blood supply of the part treated. If the Roentgen rays are continued, an obliterating endarteritis is produced. With reference to our three fundamentals, in the light of all the evidence accumulated, it would seem feasible to conclude that primarily we change a suitably prepared soil to an unsuitably prepared soil by diminishing the blood supply of the part treated. In proportion as we are able to change suitable preparation of the



soil, in like proportion are we able to destroy existing micro-organisms which require a certain type of soil for their growth. In proportion as we influence the blood supply of the lymphoid tissue in the upper respiratory tract, in like proportion are we able to influence the size of the lymphoid tissue.

#### GENERAL OBSERVATIONS.

It would seem from the foregoing that there is not anything very wonderful about the use of the Roentgen ray in certain types of impaired hearing. For years we have recognized the influence of throat conditions upon impaired hearing. In times past, we have endeavored to influence these conditions by local treatment and operative procedures. By removal of tonsils, adenoids and other tissue we have endeavored to reduce the amount of suitable soil in the upper respiratory tract. By the use of local applications we have endeavored to change a suitably prepared soil into an unsuitably prepared soil. By various kinds of local treatment intended for home use, we have endeavored to maintain this change. Through experience, we have come to recognize the influence of all this on certain forms of impaired hearing. If in the past we have secured results by adherence to these fundamentals, is it surprising that in proportion as we adhere to them when using the Roentgen ray, in like proportion do we secure results with which we are familiar. If one does very much research work, there always seem to be problems on hand and surrounding these problems questions seeking an answer. Some of the questions arising at the beginning of the work may be answered at the present time as follows:

1. Do we influence impaired hearing by means of Roentgen ray therapy?

Answer: If we have a case of impaired hearing with absence of lymphoid nodules on the posterior pharyngeal wall, open eustachian tubes, then no improvement in hearing seems to be obtained from Roentgen ray therapy. Given a case of impaired hearing with lymphoid nodules on the posterior pharyngeal wall and prominent bands of lymphoid tissue running up either side of the pharynx, just posterior to the posterior tonsillar pillar, then one seems to be able to improve the hearing in proportion as the throat condition is influenced.

2. Do we influence the hearing, or just symptoms associated with the impaired hearing?

Answer: The evidence gathered on this point seems to show that we influence head noises and the stuffy feeling in the ears. Patients gaining relief from these two symptoms are apt to state that they hear better because their head feels better.

3. When the patient states that the hearing is improved, is it possible to demonstrate this by ordinary tests?

Answer: So far, I have been unable to convince myself that it is possible to demonstrate improvement in hearing by ordinary office hearing tests. The improvement, if gained, seems to be subjective, the examiner being obliged to depend upon the patient's statements in order to know whether improvement has taken place.

4. In favorable types of cases, how long may results if secured be expected to last?

Answer: If, for sake of illustration, one cares to divide cases of chronic catarrhal otitis media into incipient, moderately advanced and far advanced type, as we classify pulmonary tuberculosis, then in incipient cases, in the lymphoid type individuals, results have been noted to last nearly two years. In moderately advanced cases, the results seem to last from three to six months, and in far advanced cases there seems to be no improvement. In the majority of cases, the first head cold following the Roentgen ray treatment seems to undo nearly all results obtained.

It hardly seems as if the use of the Roentgen ray can ever be more than an adjunct in the treatment of aural disease. I would not care to undertake the management of a case of chronic catarrhal otitis media with Roentgen ray therapy as the only means of attack. I hardly ever use it at the first office visit, preferring rather to make a general survey of the case and to institute measures, both general and local, that time has proven to be of value. But when at subsequent visits, in certain types of patients, the means instituted fail to influence a closed eustachian tube, head noises, and a more or less constant catarrhal discharge from the throat, then there comes to one a feeling of being more efficient when it is possible to influence these conditions in certain types of individ-

uals by means of small doses of the Roentgen rays. Most of you reading this have probably had more experience in the treatment of aural disease than I have had. It seems to me the only thing I can do is to relate these observations, which have come to light during an investigation of the granite cutting industry, for undoubtedly each one of you will correlate what has been written with your own previous experience and undoubtedly draw your own conclusions. To borrow a phrase from our president's address of last year, if one works on the up grade in medicine then one has to learn to labor and to wait, for time seems to be the one factor that shows us all whether our conclusions are correct.

#### SUMMARY.

1. By means of small doses of Roentgen rays we seem to diminish the blood supply of that part of the upper respiratory tract treated.

2. In proportion as we are able to diminish the blood supply of the part treated, in like proportion do we seem to cause a destruction of existing microorganisms and a diminution in the size of lymphoid tissue.

3. As the throat condition is improved, in like amount there seems to be an improvement of the aural condition.

#### BIBLIOGRAPHY.

1. Hoffman, F. L.: The Problem of Dust Phthisis in the Granite Stone Industry, U. S. Bureau of Labor Statistics Bul. No. 293.
2. Katz, S. H., and Trostel, L. J.: Dustiness of Air in Granite Cutting Plants. *Journal of the American Society of Heating and Ventilating Engineers*, 1922, No. 4, 421-442.
3. Insley, Herbert: Petrography of Rock Dust in Barre Granite Plants. *Journal of the American Society of Heating and Ventilating Engineers*, 1922, No. 4, 442-447.
4. Gardner, L. U.: *American Review of Tuberculosis*, 1920, x, 734.
5. Appendix, G.: The Problem of Dust Phthisis in the Granite Stone Industry. Bulletin No. 293 of the Bureau of Labor Statistics, United States Department of Labor.
6. Jarvis, D. C.: A Roentgen Ray Study of Dust Inhalation in the Granite Industry. *The American Journal of Roentgenology*, Vol. viii, No. 5, May, 1921, pages 244-258.
7. Jarvis, D. C.: Dunham's Fans in a Roentgen Ray Study of Granite Dust Inhalation. *The American Journal of Roentgenology*, Vol. viii, No. 10, pages 560-564.

8. Jarvis, D. C.: A Conception of Chest X-ray Densities Based on a Study of Granite Dust Inhalation. *The American Journal of Roentgenology*, Vol. ix, No. 4, April, 1922, pages 226-234.

9. Jarvis, D. C.: A Clinical Study of Granite Dust Inhalation. Transactions of the seventeenth annual meeting of the National Tuberculosis Association.

10. Jarvis, D. C.: The Upper Respiratory Tract in Granite Dust Inhalation. Transactions of the American Laryngological, Rhinological and Otological Society, 1922.

11. Witherbee, W. D.: *New York State J. Med.*, 21, Jan. 14, 1921.

12. Lederer, F. L.: The Roentgen Ray in Tonsillar Disease. *J. A. M. A.*, 79:1130, Sept. 30, 1922.

13. Babcock, James W.: Observations on the Results of Roentgen Therapy in Chronic Tonsillitis. *J. A. M. A.*, 80:300, February 3, 1923.

14. Dunham, Kennon: Treatment of Carbuncles by the Roentgen Ray. *The American Journal of Roentgenology*, May, 1916.

15. Hickey, Preston M.: Treatment of Diphtheria Carriers by Means of the Roentgen Ray. *The American Journal of Roentgenology*, 1922, ix, 319-322.

LXXIX.

OTITIS MEDIA IN SCARLET FEVER.

BY ELLISON L. ROSS, PH. D., M. D.,

FROM THE JOHN M'CORMICK INSTITUTE FOR INFECTIOUS DISEASES AND NORTHWESTERN UNIVERSITY MEDICAL SCHOOL,

CHICAGO.

The medical literature of the last sixty years has emphasized the importance of otitis media as a serious complication of scarlet fever. During that time many reports have been made of this complication. Table 1 includes the figures of the majority of these reports. This table presents data on many epidemics in many parts of the world, during a period of many years, and under the observation of many different physicians. Therefore a considerable variation is to be expected. The incidence of otitis media, as observed by different authorities, varied from 3.75 to 25 per cent of all cases of scarlet fever. Of the twelve observers, seven reported between 10 and 15 per cent, and two less than 10 per cent as the frequency of otitis media in this disease. Many of the authors specifically stated that the percentage varied greatly with the character of the epidemic.

A number of physicians during the last fifty years have studied the frequency with which scarlet fever is the causative factor of chronic suppurative otitis media. Table 2 includes all the complete data that could be found on this phase of the subject. From these data it must be said that approximately 4 to 15 per cent of all cases of chronic suppurative otitis media is due to scarlet fever. In this table the report of May is on 5,613 deafmutes and the cases are not purely chronic suppurative otitis media. It is striking to note that 10 per cent of these thousands of cases of complete deafness was due to scarlet fever.

At the Durand Hospital of the John McCormick Institute for Infectious Diseases, during the five years, 1912 to 1917, 1,305 cases of scarlet fever were cared for, and 211, or

16.17 per cent, developed otitis media. In the five years following, 1,290 cases of scarlet fever were cared for, and 159 cases of otitis media occurred, or 12.3 per cent of all that had the disease. These figures agree very well with those of the majority of the writers quoted. It is an interesting fact that the history of this complication of scarlet fever does not show any noticeable decrease with the development of more recent medicine.

Still the treatment today is practically identical with that of the earliest recorded in modern times. There were individual variations then as there are now, but treatment in general was symptomatic.

Batut<sup>14</sup> in 1886 recommended ice bag for the neck; the painting of the area about the ear with  $12\frac{1}{2}$  per cent iodoform in vaseline and the opening of the drum if pain persisted. Campbell<sup>15</sup> in 1888 used aconite as a sedative and antipyretic, leeches near the tragus and hot aural douches. Thompson<sup>16</sup> in 1890 treated the throat with a vaseline spray containing a little iodoform, refrained from inflation of the eustachian tube, used leeches, warm irrigations and advised against opening of the drum except by an expert. Jarecky<sup>17</sup> in 1905 used hot irrigations for the ear and also for the nose and throat, early paracentesis and irrigation with bichloride solution, 1:5000. He urged the removal of diseased tonsils and adenoids in all healthy children as a prophylactic against ear trouble in case of contracting scarlet fever. Yearsley<sup>2</sup> in 1909 repeated the prophylactic measures of Jarecky. He also urged frequent examination of ears of those suffering with scarlet fever independent of any ear trouble. He recommended antiseptic solutions for nose and throat washes. He quoted Gordon in the Annual Report for 1907 of the Monsell Fever Hospital, Manchester, as stating that the discontinuance of douching the nose and throat with sterile water in cases of scarlet fever increased the occurrence of otitis media from 19.7 to 20.4 per cent, and that the incidence of rhinitis increased 300 per cent. Gordon used heat, leeches, incision and irrigation with perchloride 1:1000 or 2000. Irrigation of the ear with this solution he claimed cured 84.5 per cent of all cases of acute suppurative otitis media. Fowler<sup>4</sup> used calomel, hot drinks, Dover's

powder, and bichloride 1:5000 or warm boric aural douche for acute otitis media. Alderton<sup>13</sup> in 1910 considered nose and throat medication as prophylactic treatment of little value and advised early incision and syringing with warm water or weak antiseptic solutions. Gardner<sup>18</sup> tried removing tonsils and adenoids as soon as the patient was considered fit for operation even though still suffering from scarlet fever. On the average the ears dried up in fourteen days, and the treatment never failed to dry up a primary otorrhea. The treatment with few variations followed at the Durand Hospital was to irrigate the early profusely discharging ear with warm boric acid solution and cover the ear with a gauze dressing. As the discharge became less the ear was treated with boric acid in alcohol increasing in strength from 30 to 95 per cent. The last discharges were eliminated through a gauze wick placed in the aural canal. Discharges with a foul odor were treated with weak permanganate irrigations.

There seems to be considerable difference of opinion as to etiologic mechanics of otitis media in scarlet fever. Biedert<sup>5</sup> believed that the otitis media was due to extension of infection through the eustachian tube from the throat in most, if not in all cases. He states that the ear discharges contain various organisms, such as streptococcus, staphylococcus and Klebs-Loeffler bacillus. This statement argues in his favor. Yearsley<sup>2</sup> states that the discharge from otitis media in scarlet fever contained staphylococcus aureus and albus and pneumococcus. He and Gordon, whom he quoted, direct their attention to the throat and nose, apparently on the hypothesis that the otitis is caused by infection from these places. Jarecky<sup>17</sup> stated that streptococcus pyogenes is the most common organism in the ear discharges. Batut<sup>14</sup> agreed with Biedert in that he thought that the otitis media was due to infection from the respiratory passages.

On the other hand, Alderton<sup>13</sup> stated, "In many cases examined postmortem, the normal condition of the cartilaginous eustachian tube lent strong probability to the view that the disease was a local manifestation of the general infection and not an extension of the infectious process to the ear from the throat." He stated that von Gaessler examined twelve

fatal cases of scarlet fever and found the middle ear involved in all cases, while in but one was the cartilaginous eustachian tube involved. At the Durand Hospital *streptococcus hemolyticus* has been found in every ear discharge produced by scarlet fever.

To the author it seems that the argument is in favor of considering that otitis media is primarily a result of the scarlet fever in the middle ear, but a condition often aggravated by secondary infection through extension from the throat along the eustachian tube. Weil<sup>12</sup> stated that the otitis usually occurred at the desquamation period.

Rapid and severe desquamation in the middle ear might very readily result in an irritation, as of a foreign body irritating freshly denuded walls in that location. If at this time there existed in an individual case any malformations or deformities or chronic infection in the upper respiratory tract or eustachian tube the middle ear would very readily become infected. The opinion of Dunlap<sup>18</sup> that most scarlet fever cases have a mastoiditis, supports the hypothesis that secondary infection in the middle ear is not necessary for the otitis media of scarlet fever. Gordon<sup>19</sup> states that in postmortem examinations pus was repeatedly found in mastoids where there had been no otorrhea. The lack of the usual findings of mastoiditis in scarlet fever is emphasized by the report of Borden<sup>7</sup> of 59 autopsies on scarlet fever cases, in which there was a mastoiditis in all cases and in which only six or eight cases were diagnosed before death as having mastoiditis. It is interesting at this point to note that in the otitis media of scarlet fever erosion of soft parts may be carried to the extreme, as shown by the reports of May,<sup>20</sup> in which a hemorrhage from the internal carotid artery was described, and Haynes,<sup>21</sup> in which a fatal hemorrhage from the external ear occurred.

It is often a question in connection with the otitis media of scarlet fever whether the otitis media induces other complications in the patient, and whether other complications may cause otitis media. To gather evidence on this point, we investigated the occurrence of the various complications of scarlet fever in the 2,595 cases that were cared for in the Durand Hospital in the past ten years.



In Tables 3 and 4 the number of cases of scarlet fever suffering with the commoner complications of scarlet fever, such as otitis media, cervical adenitis, nephritis, myocarditis, ulcerative tonsillitis, mastoiditis, facial paralysis and arthritis are given, with the percentage of occurrence of each. As noted elsewhere, in the first five years 16 per cent developed otitis media, and in the second five years 12 per cent, which harmonizes well with the findings of others. In the first five years data were collected on cervical adenitis of all grades, and it was found that 89 per cent of the patients had some degree of this condition. During the second five years cervical adenitis only of a severe sort was noted, and this occurred in 30 per cent of the cases. Nephritis during the first five years affected about 12 per cent and, during the second five years, about 6 per cent of the scarlet fever patients. Myocarditis in the first period was found in 7 per cent of the cases, but in the second period in about 1 per cent. Ulcerative tonsillitis varied from the first period with less than .2 per cent to the second period with about 4 per cent. Mastoiditis occurred in about 1 per cent of the cases in both five year periods. Facial paralysis was rare. Arthritis, one of the most important complications of scarlet fever, was found present in about 12 per cent of the cases in the first period and 9 per cent of the cases in the last period.

We have considered that if the percentage of a complication without otitis media is small and the percentage of the complication with otitis media is large, the evidence is strong that the complication was contributed to by otitis media, independent of the percentage of occurrence of the complication among the total number of cases with otitis media. For example, let us start with the hypothesis that practically all ruptured ear drums are the result of otitis media. The percentage of ruptured ear drums with no otitis media would be small, and the percentage of ruptured ear drums with otitis media would be large, while the percentage of cases of otitis media with ruptured ear drums might be any number and not alter our conclusions. In studying Tables 5 and 6, we find no such argument in favor of otitis media having contributed to any other complication except the very obvious complications of mastoiditis and facial paralysis.

Using the same system of reasoning as in the previous paragraph, evidence of any complication contributing to the production of otitis media would be shown in Tables 5 and 6 by a small percentage of otitis media without the complication and a large percentage of otitis media with the complication, independent of the percentage of the complication with otitis media. If the figure in the first column in the percentage tables is near 100 and the one in the last column is near zero, there is evidence of the corresponding complication contributing to the production of otitis media. In studying these tables, we find no evidence than any of the listed complications predispose the patient to otitis media. This was rather surprising in the case of ulcerative tonsillitis.

The author is much indebted to Dr. Ludwig Hektoen and Dr. George H. Weaver for their assistance in this piece of work.

#### SUMMARY AND CONCLUSIONS.

The 2,595 cases of scarlet fever cared for in the Durand Hospital developed acute suppurative otitis media in 16 and 12 per cent in the first and second five years, respectively.

Of the complications studied, nephritis, myocarditis, ulcerative tonsillitis and arthritis, none of them appeared to be responsible for the otitis media, and the otitis media did not predispose the patient to the complications named.

TABLE 1.

Occurrence of otitis media in scarlet fever.

Authors	Reference	No. Cases Scarlet Fever	No. Cases Otitis Media	Pct. O. M. in Scarl. F.
LeMarch'hadour .....	1	339	36	10.65
Yearsley .....	2	17829	2355	13.20
Sprague-Bezold .....	3	17087	64	3.75
Fowler-Baeder .....	4	51	13	25.00
Biedert .....	5	25882	3833	14.80
Biedert-Finley .....	5	4397	439	10.00
Biedert-Craiger .....	5	1008	131	13.00
Nager .....	6	744	119	16.00
Borden .....	7	746	65	8.70
Holmgren .....	8	9590	2020	21.00
Alderton .....	13	1621	178	11.00
McCrae .....	22	850	125	14.70

TABLE 2.

Scarlet fever as a cause of chronic suppurative otitis media.

Authors	Reference	No. Cases Otitis Media	No. Cases Due Scarlet Fever	Pct. Ot. Due to Scarl. F.
May-Schmalz .....	9	2500	137	5.50
May-Yearsley .....	9	544	27	5.00
May-Burkhardt-Marian .....	9	1950	85	4.33
May .....	9	5613 D.M.	572	10.18
Bezold .....	10	623	89	14.30
Bezold .....	11	714	103	14.40
Sprague-Blau et al. ....	3	14045	55	3.90
Weil-Burkhardt .....	12	4309	488	11.30

TABLE 3.

Complications of scarlet fever. First five years.

	Total	With Otitis Media	Without Otitis Media	Percent. Total Cases (1305)
Otitis Media .....	211			16.17
Adenitis; all grades.....	1166	201	965	89.35
Nephritis .....	151	48	103	11.57
Myocarditis .....	92	23	69	7.05
Ulcerative Tonsillitis.....	2	0	2	0.15
Mastoiditis .....	16	16	0	1.23
Facial Paralysis.....	0	0	0	0.00
Arthritis .....	155	37	118	11.88

TABLE 5.

Complications of scarlet fever in relation to otitis media—expressed in per cent.

	Per cent Otitis Media with Following	Per cent of Following with Otitis Media	Per cent Fol- lowing without Otitis Media
Adenitis; all grades.....	95.27	17.24	82.76
Nephritis .....	22.85	31.79	68.21
Myocarditis .....	10.90	25.00	75.00
Ulcerative Tonsillitis.....	0.00	0.00	100.00
Mastoiditis .....	7.58	100.00	0.00
Facial Paralysis.....	0.00	0.00	0.00
Arthritis .....	17.54	23.87	76.13

TABLE 6.

Other complications of scarlet fever in relation to otitis media—expressed in per cent.

	Per cent Otitis Media with Following	Per cent of Following with Otitis Media	Per cent Fol- lowing without Otitis Media
Adenitis, C. Severe.....	44.7	18.4	81.6
Nephritis .....	11.9	25.7	74.3
Myocarditis .....	0.6	5.5	94.5
Ulcerative Tonsillitis.....	6.9	23.9	76.1
Mastoiditis .....	7.5	92.3	7.7
Facial Paralysis.....	2.5	100.0	0.0
Arthritis .....	10.7	14.7	85.3

TABLE 4.

Other complications of scarlet fever. Second five years.

	Total	With Otitis Media	Without Otitis Media	Per cent Total (1290) with Following
Otitis Media.....	159	.....	.....	12.3
Adenitis, C. Severe.....	385	71	314	29.8
Nephritis.....	74	19	55	5.7
Myocarditis.....	18	1	17	1.4
Ulcerative Tonsillitis.....	46	11	35	3.6
Mastoiditis.....	13	12	1	1.0
Facial Paralysis.....	4	4	0	0.4
Arthritis.....	116	17	99	9.0

## BIBLIOGRAPHY.

1. Le March'hadour: Ann. de mal. de l'oreille du larynx, XXIX, 1903, p. 455.
2. Yearsley, M.: Practitioner, London, 1909, page 88.
3. Sprague, F. B.: Amer. J. M. Sc., Phila. and N. Y., 1905, CXXX, 423.
4. Fowler, E. P.: Amer. J. Obst., N. Y., 1908, LVIII, 530.
5. Biedert, C. C.: Amer. Med., Phila., 1906, N. S., I, 210.
6. Nager, F. R.: Cor-Bl. f. schw. Aerzte, Basel, 1908, XXXVIII, 593.
7. Borden, C. R. C.: Boston M. and S. J., 1913, CLXVIII.
8. Holmgren, Gunnar: Arch. f. Ohrenheilk., 1912, XC, 52.
9. May, C. H.: Amer. J. Obst., N. Y., 1889, 362.
10. Bezold, F.: Arch. f. Ohrenheilk., Leipzig, 1884, XXI, 251.
11. Bezold, F.: Arch. f. Ohrenheilk., Leipzig, 1887, XXVI, 231.
12. Well, A. J.: N. Orl. Med. and Surg. J., 1911, LXIV, 210.
13. Alderton, W. A.: Tr. Amer. Laryngol., Rhinol. and Otol. Soc., 1910, 227.
14. Batut, L.: Arch. de Med. et Pharm. Mil., Par., 1886, VIII, 266-359.
15. Campbell, D. M.: Physician and Surgeon, Ann Arbor, Mich., 1888, X, 193.
16. Thompson, J. H.: Med. Mirror, St. Louis, 1890, I, 65-68.
- 17.
18. Dunlap, A. M.: Laryngoscope, 1922, XXXII, 272.
19. Gordan, A. K.: Brit. J. Nursing, London, 1906, XXXVII, 23.
20. May, B.: Annals Surgery, St. Louis, 1885, I, 559.
21. Hynes, P. J.: Lancet, London, 1870, II, 431.
22. McCrae, John: Can. Med. Assoc. J., April and May, 1911, 293.

HEMORRHAGE FOLLOWING THE ADENOID  
OPERATION.\*

BY FRED W. BAILEY, M. S., M. D., F. A. C. S.,

CEDAR RAPIDS, IOWA.

The operation for the removal of adenoids is perhaps performed more often than any other operation excepting that for the removal of the faucial tonsils. Due to the fact that in most cases the adenoid tissue of the pharynx atrophies beginning about the sixteenth year, the operation for the removal of adenoids is performed mostly in children.

The adenoid tissue is located in the nasopharynx and is very vascular. The blood supply of the adenoid is derived from branches of the ascending and descending palatine artery and from branches derived from the tonsillar artery. There may, of course, be anomalous vessels present in this region, just as in any other region of the body.

Surgical removal of the adenoids is always accompanied by more or less hemorrhage. The hemorrhage usually is very profuse for a few seconds and then gradually ceases. As a general rule, no attention is given to the control of the hemorrhage at the time of operation and the patient is allowed to bleed until the bleeding stops. Occasionally, however, there is a case where the hemorrhage does not stop as soon as one would reasonably expect, or where it occurs some time after the patient has been removed from the operating table, the time varying from an hour or so to a few days or more.

It is on this occasional case of hemorrhage that I wish to direct a few remarks in this paper.

In looking into the subject of hemorrhage following the adenoid operation, I picked up at random seven textbooks from the shelves of a medical library, all these books written by teachers and authorities of recognized ability. Only one

\*Read before the Sioux Valley Academy of Ophthalmology and Otolaryngology, January 24, 1922.

of the seven spoke of any method of controlling the hemorrhage from the adenoid region other than by direct pressure. This one authority advises picking up and pinching off the bleeding points to be sure that all hemorrhage is controlled before the patient leaves the table.

This authority, Doctor Beck, advises retracting the palate by means of a rubber catheter introduced through the nose, bringing into view the region from which the adenoid tissue has been removed. A number of the authorities referred to the possibility of a secondary hemorrhage and advised the introduction of a posterior tampon in case the hemorrhage did not stop.

In looking up mortalities following operation for an adenoid which might be attributed to hemorrhage, one finds very little recorded. One authority reports ten cases of death following this operation, and one often hears of an occasional death or reads the account of one in some journal. In a series of twenty-five hundred adenoid operations that I have performed in the past ten years, I have never had a death following this operation, and have had only one severe secondary and one severe primary or rather reactionary hemorrhage following this operation.

This secondary hemorrhage occurred in a lad five years old upon whom I had operated for the removal of tonsils and adenoids on October 5, 1918. This was at the time of the epidemic of influenza, and it happened that on the fifth day when the little patient began to bleed in an alarming manner, I myself was in bed with influenza and pneumonia, so my assistant took care of the case. He reported that the little lad was bleeding very severely and that he was unable to stop it, and I advised that he plug the posterior nasal pharynx, which he did. Even with this tampon in, the youngster would bleed from time to time, and before the bleeding was entirely stopped he had lost a great deal of blood and was in rather a serious condition. The administration of thromboplastin in this case did not seem to do much good. The next day after the bleeding stopped, the little patient came down with a severe attack of influenza, and it may have been that this trouble coming on had something to do with the secondary hemorrhage occurring.

The case of primary or rather reactionary hemorrhage that I encountered is the one I wish especially to deal with. This happened in a boy age nine, upon whom I operated for tonsils and adenoids September 16, 1922. A coagulation test in this case came back with the report of three minutes. I operated on the patient about 8 o'clock in the morning for the removal of adenoids and tonsils. About 11 o'clock the patient began to bleed profusely through the nose and mouth. An assistant attempted to stop the hemorrhage by means of pressure in the tonsillar fossæ, thinking that the hemorrhage must necessarily come from this place. The bleeding stopped somewhat, and when I saw the patient, about 12 o'clock, had almost ceased. The patient, however, was very white and the pulse weak and rapid. At 12:30, I was called back to the hospital, the nurse reporting that the patient was bleeding very severely again. I was unable to examine the throat satisfactorily, so I had the patient removed to the operating room and a little anesthesia given, thinking that I had a tonsillar hemorrhage to deal with. However, after I had cleared the clots from the tonsillar fossæ, I found them perfectly dry and could see blood coming from back of the palate from the region of the adenoids. I cleared out the clots from this region by means of the forceps and Beck suction apparatus and, by retracting the palate, could see that the hemorrhage was from this region. I at first attempted to control it by grasping the bleeding point and crushing it with the angiotribe. This was not successful, and I thought I would put in a posterior tampon and, in fact, had one brought in, when I hit upon the idea of putting a suture around the bleeding point just as I had been in the habit of doing in cases of hemorrhage from the tonsils. I used No. 0 catgut and a modified Ingersol tonsil needle and had no trouble in putting a suture around the bleeding point, and had the satisfaction of seeing the hemorrhage stop at once as soon as the suture was tied. The patient was removed to his bed and made an uneventful recovery.

Since that experience I have used sutures in three other cases in which the adenoids bled more than I thought ordinary. I had no trouble finding the bleeding point and no trouble introducing the suture, and the bleeding stopped at once and did not recur.

In conclusion, I wish to emphasize the advisability of conserving blood during the operation for the removal of adenoids. This operation is usually done in connection with the removal of tonsils. It is not good surgical judgment to allow a patient to lose more blood than necessary. A properly chosen piece of gauze applied by means of the hemostat and pressed firmly into the adenoid region will control the hemorrhage and prevent the patient from losing very much blood during this operation. If this does not succeed, the palate can be retracted and the bleeding points merely picked up with a long Elliot forceps, and this alone will usually stop the hemorrhage. I do not believe the picking up and crushing of the bleeding points in either the adenoid or tonsil operation to be good judgment, as death of tissue and sloughing must necessarily occur and a fertile field for invading pathogenic organisms is created. Also, one is not sure by this method that the bleeding will stay stopped, as is often demonstrated when this method is used in stopping tonsillar hemorrhage. A suture in the proper place seems to me the logical method to attempt to control an undue adenoid hemorrhage. It is easy to apply, certain in its results and no damage is done.

SUITE 308-9-10-11-12 SECURITY BANK BLDG.



## SOCALLED PRIMARY THROMBOSIS OF THE LATERAL SINUS, WITH PRESENTATION OF AN INTERESTING CASE.

BY E. T. SENSENEY, A. B., M. D.,

ST. LOUIS.

When one considers the pathology of so-called primary thrombosis of the lateral sinus, he is forced to conclude that, with rare and doubtful exceptions, such a condition does not exist. Thrombosis is divided into two definite classes, medical and surgical. Bertier<sup>1</sup> defines a surgical thrombus as one due to some local cause, suppurative in nature and limited in extent, while a medical thrombus is one which arises from some general blood state or causes, acting from a distance, nonsuppurative and widespread in character. Severe chlorosis is mentioned as such a cause.

An interesting medical case is reported by F. R. Sims.<sup>2</sup> A man, 49 years of age, in supposedly excellent health, after attempting to suppress a sneeze, felt that something had given away in the left side of his head. A few minutes later he complained of severe headache, chiefly confined to the left side. This headache was accompanied by severe nausea and vomiting, which persisted for twelve hours without relief. His temperature was normal. Pulse, 96. He later developed various neurologic symptoms, necessitating his commitment to a hospital for the insane, where he died of bronchopneumonia two and one-half months later. The case was carefully studied during life, and the most probable diagnoses, brain tumor, apoplexy and meningitis, were excluded. The true nature of the disease was disclosed by autopsy, when a thrombus was found filling the left sigmoid and lateral sinuses. The probable cause was explained by the presence of arteriosclerotic vessels in the sinus wall. Sims thinks that "a weak vessel gave way under increased blood pressure and a small hemorrhage occurred. This led to other hemorrhages,

starting a vicious circle, similar to the condition found in internal hemorrhagic pachymeningitis. The fresh hemorrhages in the thrombus help to confirm this idea."

Exception is taken by E. Therman<sup>3</sup> to the term "primary," even in these medical cases. He believes that in the majority, if not in all, the thrombosis is secondary to inflammatory changes about the sinus or in the sinus wall. He bases his conclusions both on the experimental production of thrombosis in rabbits and on the careful microscopic pathologic study of four cases coming to autopsy. The etiologic factor in one case was psychosis, in one luetic arteriosclerosis, in one tuberculosis, and the fourth arose in the course of acute endometritis following miscarriage. In spite of these various causes, he found in each case inflammatory changes in the meninges and brain of two kinds, one more or less chronic and the other acute.

The diseased blood changes and local disturbances of circulation to which many authors attach great importance in the determination of the origin of a thrombus are, in the opinion of Therman, to be considered rather as consequences of infection and inflammation than as real causative factors. He states that it is a well known fact that young people suffering from chlorosis and anemia are, at the onset of an infectious disease, more disposed to encephalitis and sinus thrombosis. His experiments in the artificial production of thrombosis in rabbits show, first, that it is difficult to produce sinus thrombosis in a mechanical way, and second, that the injection of chemically acting substances into the blood vessels to create a thrombus also develops an inflammatory process in the vessel walls and in the surrounding tissues.

All of which has some bearing on our understanding of the pathology of surgical thrombosis. We may accept the fact that we must have an inflammatory or infectious process contiguous to, and in the walls of the vein, whether this infection occurs by metastasis from more or less distant source, or by direct extension from the mastoid to the sigmoid sinus, or from the middle ear to the jugular bulb. Don. R. Campbell<sup>4</sup> states very concisely the ways in which this extension may take place, as follows:

"(a) The septic inflammatory process may, in the first place, proceed by a rapidly widening area of involvement, without the formation of a protective process limiting the advance of the infection. In this type there is little effort to throw up a limiting protective plastic area around the area of infection, and the process proceeds rapidly by open venous channels, which quickly carry the infection to outlying areas.

"In this type the inner surface of the bone is quickly reached without the intervening osseous area having undergone any great amount of caries or necrosis, so that an infection of the walls of the venous channels takes place without macroscopic evidence of bone destruction, furnishing the type of lateral sinus thrombosis occurring in acute tympanomastoiditis.

"(b) On the other hand, there is that type of septic process characterized by the throwing up of a barrier to the progress of the infection, which more or less effectually limits its onward course.

"In this type, thrombosis takes place in the venules of the bone involved in the protective area, cutting off its nutrition, but limiting the progress of the infection; then instead of a rapidly widening area of infection with intact bony structure, as in the former process, in this type the nutrition of the bone suffers, caries takes place, and the picture of a breached internal plate, with perhaps an extradural abscess, is presented.

"These are cases which, in the primary destructive bone processes, are of that type in which the onward progress of the infection has been stayed by the thrombosis in the venules of the bone, and the force of the infection spent on the bone tissue itself, the spread to the soft tissues further on having been checked by the venous bone thrombosis.

"In such a case the status of an original acute mastoid disease is brought about by a secondary infection which now might easily extend to the vein, causing phlebitis and subsequent thrombosis of its contents.

"The picture one must carry of the events leading to a thrombosis of the blood in the vessel must, in its early stages, be that of a phlebitis, an infection of the tissues making up the walls of the vein, the inner lining at first remaining intact, and the contained blood passing on uninfected."

We must also remember that there are certain bacteria which may produce intracranial complications after the otitis media is entirely healed. Important among these are the encapsulated bacteria—e. g., streptococcus mucosus and pneumococcus. Furthermore, the streptococcus mucosus sometimes produces an otitis media and mastoiditis, in which the only symptoms are slight tinnitus or feeling of fullness, which may be readily overlooked. With these facts in mind one may, in looking over the literature, explain most of the cases reported as "primary" sinus thrombosis.

An interesting case is that reported by Samuel Lodge, Jr.<sup>4</sup> A strong, healthy man of 59 years gave no history of syphilis, no history of injury to head or ear, no rheumatism, no history of discharge from the ears. For six months this patient had complained of deafness, and often pain, in the right ear. Gradually the attacks of pain became more frequent, and there was also a loud noise in his ear. He finally developed a boggy edematous area over and beyond the right mastoid region. The drum membrane showed no signs of old perforation or important gross changes. Mastoid operation showed no macroscopic pathology. The patient improved rapidly, and seemed quite well until the twentieth day following operation, when he became semiconscious and developed a septic temperature with chills. Pupils equal, no deviation of the eyes, no retraction of the head, no localizing symptoms. In further operation, the lateral sinus could not be found, there was no extradural abscess, and exploratory search revealed no temporosphenoidal abscess. The patient died the next day. Partial autopsy showed a meningitis and encephalitis. The left lateral sinus was filled with a thrombus, extending from the torcula to the sigmoid portion. The right lateral sinus was obliterated, its walls adherent, and the bony groove almost obliterated. This condition existed from the torcula to the bulb, and the internal jugular was a threadlike cord. The history excluded a marasmic thrombus. There are no bacteriologic or microscopic notes. The author considers that "the cause of death in this case was presumably due to an extension thrombus forming in the left lateral sinus, thus throwing a greater strain on the collateral intracranial venous circulation than it could bear, on account of the right jugular and lateral

sinus being already occluded." The report of the Clinical Research Association, to which the thrombus was sent, states that "the thrombus is undoubtedly antemortem in formation." Interrogation of the patient's wife brought forth the information that fifteen years previous to his death, he had suffered from a severe neuralgia on the right side of his neck and head. The author believes that the obliterative process began at that time, and continued until the left lateral sinus became involved. His ear was not affected at that time.

This report was published in 1900. From the scanty evidence, it is hard to deduce just when the obliterative process in the right lateral sinus commenced. The case is suggestive of streptococcus mucosus. An obliterative process in the right jugular and right lateral sinus most certainly would not have produced deafness in the right ear. It seems quite possible that the whole process commenced coincident with the pain and deafness, seven months prior to the exodus. Therman's<sup>8</sup> findings in his medical cases seem to hold good in this case, since inflammatory changes were present in the brain and meninges.

Thrombus of the internal jugular vein may follow a severe tonsillitis or a septic process in the throat and neck. Goodman<sup>5</sup> and Long<sup>6</sup> both have reported such cases. Lynah<sup>7</sup> has reported a case of internal jugular thrombosis following a septic (streptococcus) process in the throat and neck, in which autopsy showed an extension thrombus, septic in character, in the sigmoid portion of the lateral sinus.

The case which I wish to present is interesting because it followed a septic process in the throat, presented some difficulty in diagnosis, and offers some interesting pathologic deductions. Mr. H., a young man of 25 years, healthy, vigorous, with no previous history of moment, was referred to me three weeks following a severe throat infection on the right side. For three days prior to my examination, May 19, 1920, he had chills and high fever, accompanied by right sided headache. He claimed that never in his life had he had any ear trouble whatsoever, and that during his illness his ear never hurt him at any time, nor was there any discharge from it. At the time I examined him his hearing was normal. The

membrana tympani showed nothing abnormal; there was no mastoid tenderness or tenderness posterior to the mastoid. There was swelling and induration in the right side of his neck, and several cervical glands could be distinctly palpated. The neck was very sensitive to pressure, and pressure over the jugular gave pain. Temperature, day of admission to the hospital, 100° F. to 104.3° F. Pulse 110. Neurologic examination negative. General physical examination negative. Urine normal. Blood culture negative. No malarial plasmodia in blood. Leucocyte count, 14,400. Eyes: Veins of fundus of both eyes engorged, discs normal; normal reaction of pupils.

A tentative diagnosis of thrombosis of the internal jugular, secondary to the throat infection, was made. May 20, 1920, the right internal jugular was exposed. There were a number of infected cervical glands, some of which were removed. The internal jugular vein was bared as high up as surgically possible and for at least three inches of its course. It appeared normal, except that it seemed smaller in size than is usual. The facial vein was ligated as well as the jugular, and a portion of the jugular two inches in length was resected and opened. There was no thrombus. It was then decided to expose the lateral sinus, on account of the septic temperature which could not be explained on other grounds, and on account of the apparent small size of the jugular. The mastoid was macroscopically negative. There was no pus in the antrum or in any cell. The lining membrane did not seem to be congested. The inner plate showed no macroscopic changes. The sigmoid portion of the sinus was laid bare. There was no extradural abscess. The dura over the sinus showed no granulations, and was easily separated from the bone. The sigmoid sinus, however, from the knee to the bulb, was easily outlined, slaty blue in color, except for about one-half inch of its midportion, which was distinctly yellowish. Upon opening, pus under pressure was expelled from the abscess in this portion. An occluding thrombus was removed, extending from the knee of the sinus to the bulb; free bleeding was obtained at both ends. The resected portion of the jugular and pus from the abscess were sent to the laboratory for examination.

Temperature fell to normal immediately following the operation. The case proceeded uneventfully to recovery, and was discharged from the hospital on the tenth day.

The hospital laboratory report on the jugular was without interest. The culture from the sinus abscess, much to my chagrin, was reported as contaminated in the laboratory. I regret that the bone removed was not subjected to microscopic examination.

Two explanations are possible in this case. First, that infection of the lymphatic system of the internal jugular had extended upward, without producing changes in the jugular itself, to infect the lateral sinus, with the production of a thrombus. Second, that there was a *streptococcus mucosus* otitis media and mastoiditis, which on account of the severity of the throat symptoms, was unnoticed by the patient. That this process had infected the sinus wall through blood channels, and had itself subsided before the patient entered the hospital. The latter seems the most logical conclusion, since it is not clear why an abscess should occur so far from the primary source of infection.

#### SUMMARY.

We come to the conclusion that the term "primary" used in connection with sinus thrombosis is a misnomer. This is particularly true of the surgical cases. In a critical review of cases from 1900 to this year, we have failed to find any which could not be reasoned as secondary. In many reports, the material offered is inadequate.

We conclude that thrombosis of the lateral sinus may be secondary to:

1. An infectious or inflammatory process contiguous to the vessel wall.
2. Mastoid infection by contiguity or by extension from infected veins of the mastoid.
3. Middle ear infection of the jugular bulb and by extension to the sinus.
4. Jugular vein thrombosis and by extension to the sinus.

We feel that, in the light of these obscure cases, many individuals die with a diagnosis of septicemia, who have unrec-

ognized sinus thrombosis. And that a closer association of the internist and neurologist with the otologic surgeon would result in the clearing up of many cases presenting a septic temperature of unexplained origin.

## REFERENCES.

1. Bertier, J.: Medical Thrombosis of the Sinuses of the Dura Mater. *Arch. Gen. de Med.*, April, 1907.
2. Sims, F. R.: A Case of Autochthonous Sinus Thrombosis. *Boston M. and S. J.*, 1910, CLXIII, 218-220.
3. Therman, Ernest: Ueber die sogenannte primäre Sinus-thrombose. *Arb. a. d. Path. Inst. d. Univ. Helsingfors, Berlin*, 1907, 1, 673-683.
4. Lodge, S., Jr.: Two Cases of Thrombosis of the Lateral Sinus, in One of Which the Etiology Was Obscure. *J. Laryngology, London*, 1900, XV, 493-498.
5. Campbell, Don R.: Lateral Sinus Thrombosis. *Laryngoscope*, Oct., 1922.
6. Goodman, C.: Primary Jugular Thrombosis Due to Tonsil Infection. *Annals of O., R. and L.*, June, 1917.
7. Long, J. W.: Excision of Internal Jugular Vein for Streptococcic Thromb. *Surg., Gyn. and Obstet.*, Jan., 1912.
8. Lynah, H. L.: Primary Lateral Sinus Thrombosis Without Involvement of the Mastoid Process or Tympanum. *Annals of O., R. and L.*, Sept., 1912.

316 LISTER BUILDING.



LXXXII.

FULMINATING SEPTIC INFECTION OF THE  
PHARYNX AND LARYNX.\*

BY HARMON SMITH, M. D., Sc. D.,

NEW YORK.

In presenting this subject I am actuated largely by personal experiences, occurring particularly in the last two years, in each of which the gravity of the case was such that it was at times doubtful if the patient would recover. In one case a tracheotomy was necessary; in two it seemed almost unavoidable, and in the third the patient succumbed.

I will describe only those very grave conditions which, beginning in the pharynx, particularly in the tonsils, extend downwards towards or into the larynx. I will not include Ludwig's angina or the many types of phlegmon involving the mouth, submaxillary region, tongue, etc.

We may reasonably ask why, in an apparently simple infection of the tonsil or lateral pharyngeal wall, there will suddenly develop an extensive inflammatory infiltration of one or both sides of the pharynx, followed by edema of the uvula, possibly of the glottis, with no pus focus apparent, and this accompanied with difficult respiration, profound physical exhaustion and all the symptoms of general sepsis?

The simple beginning is nothing more than is frequently treated by the general practitioner with the administration of a salicylate and a simple gargle; nor does it at first present, even to the eye of the specialist, any unusual evidences beyond that observed by him daily in his clinic. For a possible answer, I would call your attention to the etiology of this condition as offering an explanation, which was first emphasized a number of years ago by Dr. Jonathan Wright, and which he has enlarged upon more recently at my personal request.

In the first place there exists that profound depression of the sympathetic nervous system which may have been in-

---

\*Read before the Forty-sixth Annual Congress of the American Laryngological Association, June 3, 1924.

initiated by shocks of various kinds, as an alcoholic debauch, insufficient or improper food, great mental or physical fatigue, or sudden exposure to cold. Underlying this there may also exist Bright's disease, diabetes or lithemic conditions.

These shocks may not be sudden or severe, but may be insidious and unobserved, but whatever the manifest or hidden disturbances are they bring about that unsolved state of molecular change in the ganglia of the sympathetic nervous system. Through this nervous system, some impulse is conveyed which sets up dialysis which alters the biochemical resistance at one extremity of the nerve. The peripheral termination of the nerve is in the epithelium of the tonsil or pharynx, or other parts of the buccal mucosa. The changes wrought there in the lipoid or lipin and the protolipin of the cells eventuate in a change of surface tension. The normal status previously existing there is one of balance. The streptococcus or some other organism lies along the surface of the cell, with a neutral zone between its surface and that of its host, either in a tonsil crypt or wherever it is lodged. Both surfaces, that of the cell and that of the bacterium, are integral parts of living protoplasm, and protoplasm lives by virtue of this very susceptibility to alterations by stimuli. Whenever that biophysical or biochemical change in the cholesterol or lecithin or soap and its protein links occurs, the streptococcus gains entrance. Now this invasion by the surface bacterium may occur without appreciable damage to the patient. Probably there most frequently happens that as soon as the streptococcus gets in it is destroyed by a phagocyte or some chemical within or outside the phagocyte, bringing about lysis or dissolution. Any toxin that the streptococcus may carry in meets an antitoxin which destroys its activity, and its potentiality for warfare is destroyed. However, the opposite of this hoped for fate of the streptococcus sometimes happens, and that same nerve impulse which permitted its penetration will occasion a branch impulse which annihilates the bactericidal juice of the phagocyte and paralyzes the phagocyte itself. The cellular structure, or whatever makes the antitoxin, rebels and the streptococcus is unopposed in its nefarious actions. It is the combination of these three conditions which I believe to be the cause of the type of throat to which I would call your attention. When only one

or two of these conditions occur, as, for instance, the biochemical change of surface epithelium, permitting the invasion of the streptococcus, and only the paralysis of the phagocyte results, we may get a purulent inflammation, but the antitoxin remains to guard against invasion of adjacent tissues, and the result is an ordinary quinsy which when concentrated can be easily overcome by surgical means. But when surface resistance collapses and phagocytic paralysis and inhibited phagocytic juice is combined with the defense of antitoxin annihilated, the streptococcus runs riot and no man can predict the limit of its invasion or destruction. It is only by keeping in mind that there is a multiplicity of factors in the defense of the organism and that the possible, though extremely rare combination of all at once giving way, precipitates the fulminating, appalling attack, we secure a tenable etiologic conception of these cases. The larger the number that fail in the defense and the more simultaneous their failure, the graver is the clinical picture.

Granting the truth of the argument presented, that the protective barriers enumerated have been let down and the streptococcus penetrates without resistance the tissues of the tonsil or pharynx, what happens then to the invaded structures? Ferments form which assimilate and digest the unresisting tissues, producing unexpected destruction and extension, even to the involvement of the general blood stream. The prevailing organism in this type of case has been the streptococcus hemolyticus, though mixed cultures have been found in some cases. Even in so-called healthy tonsils this type of organism is found in 50 per cent of cases, demonstrating its eternal preparedness to invade an unprotected region.

Admitting the liability to error in the foregoing premises, we can at least relate indisputable facts relative to the subjective and objective symptoms in patients infected by this organism. In all cases of this fulminating type there has been either lowered resistance from mental fatigue, physical overwork, or an underlying dyscrasia. The patient will outline a history of having had a sore throat, perhaps mild in character and having done too much in one way or another feels tired, debilitated and without "pep." These prodromal symptoms are followed by an unnatural feeling of fullness or discomfort

in one or both sides of the pharynx. This is rapidly followed by a sharp pain on swallowing or by interrupted spasms of pain in one or both tonsils. The increasing fullness, pain or discomfort leads to progressive difficulty in breathing and *malaise*, which is often followed by a chill. The subjective symptoms are so disproportionately severe, as compared to the evidence of the local trouble, that the patient immediately consults a doctor and does not employ the home remedies which have previously been resorted to in throat affections. In certain instances, where there has been some delay, the doctor finds the patient on the verge of collapse. There is difficulty in swallowing, the breathing is labored, possibly there is dyspnea, the pulse is rapid, and the temperature is relatively low for the other systemic symptoms. The anxiety of the patient is evidenced in his face; beads of perspiration appear on the brow, the lips are blue, and the picture is most alarming. Tracheotomy immediately suggests itself as the only available means at command to save the patient's life. However, this necessity does not obtain except in a limited number of cases, and the symptoms just enumerated represent only the extreme types of this rapidly fulminating phlegmon.

I will now relate a few personal experiences which will not only present the subjective and objective symptoms of this condition, but also give in detail the treatment which has been employed.

Mr. J. McC., opera singer by profession, was first seen by me for a slight laryngitis and tracheitis in February, 1922, having been referred to me by Dr. Norval Pierce, of Chicago. The examination at that time only revealed, in addition to the acute condition, a spur on the right septum and markedly enlarged tonsils. Following this examination he undertook an extended Western concert tour of an unusual, trying nature, and when passing through Chicago en route for New York City, consulted Dr. Pierce, who warned him of his lowered physical condition and overtaxed vocal exertions. On Saturday, April 8, 1922, he called at my office complaining of discomfort in his throat. His anxiety was aggravated because he was to give a concert in New York City the next evening. Both tonsils were swollen and hyperemic, but I felt certain that an astringent gargle and a purge would overcome the

condition sufficiently to enable him to meet his obligation. On Saturday morning, April 9th, however, he had a few white patches on the left tonsil, and I advised him to cancel his engagement. His temperature was only 100 by mouth and his pulse slightly accelerated. By evening of the same day, both tonsils had coalescent white patches on them, and the right, which was uninvolved in the morning, was the worse, and gave evidence of an edematous infiltration, which led me to believe he was in for a quinsy. I at once applied an ice bag externally and instituted hot saline irrigations to be followed by a tincture of guaiac gargle. On Monday, April 10th, I incised what I anticipated to be a pus pocket in the right tonsil, but only a small amount of serous fluid and blood was evacuated. Both tonsils and lateral pharyngeal walls now rapidly became infiltrated with marked edema of uvula and palate. I had already taken a culture from the tonsils, which showed a predominance of streptococcus hemolyticus with a few spirilla of Vincent, the latter probably coming from his teeth. The condition advanced rapidly, even noticeably, from hour to hour, so that by Tuesday, the 11th, both sides of the neck were swollen down to the clavicle, and the patient could hardly swallow and breathed with difficulty. Ice packs had been kept on externally, the throat irrigated continually, the bowels evacuated by catharsis and colonic irrigation, and constant attention rendered by the general practitioner and myself. A tracheotomy set was in constant readiness, and I spent my entire time day and night at the home of the patient, ready and equipped for any emergency which might arise. By April 12th, there was marked edema of the lateral walls of the pharynx below the tonsils, involving as well the epiglottis. The uvula had been so compressed between the tonsils that it assumed a pyramidal shape and almost prohibited breathing by being pressed into the laryngopharynx.

I had Dr. Coakley see him in consultation, but his efforts to find a focal pus cavity were unavailing, and he acquiesced in the method of treatment being pursued. Later I had Dr. MacKenty in consultation to see if he could pass a tube through the patient's nose and into the pharynx, by which he could get more air, but in this he was unsuccessful. Until April 17th it was necessary to incise different areas of edema to evacuate

some of the serum segregated at these points, so that the patient could breathe.

No free pus was ever evacuated, but by April 17th, the symptoms began to abate, and from then until complete recovery the cyanosis and dyspnea lessened. Knowing the psychology of the patient, I spared no effort to obviate performing a tracheotomy, but in one who was not a singer I would have done a tracheotomy on the second or third day. As the results have justified my conservatism, I feel highly gratified, but it was assuming an awful risk. I might add that he sings as well as before his experience, but if I had performed a tracheotomy his nature is one of such complexity that he not only would never have sung as well, but would never have even attempted to sing. Even as it was, I had great difficulty in inducing him to return to New York for his concert work the following autumn, as his London doctors had urgently advised him to give up work for one year, which in my opinion would have been fatal, as few singers or prize fighters ever come back.

The next case, No. 2, is one of the most interesting in my experience. Mrs. J. R. G., was referred to me by Dr. Harlow Brooks to determine if her tonsils were the cause of her arthritis. This lady, past fifty, came to me April 19, 1923. She complained of only an occasional sore throat in addition to her arthritis, which seemed to concentrate more in her fingers than elsewhere. She was a grandmother, and I felt that focal infections of the tonsils should be eliminated at her age, although she had had teeth removed and colonic irrigations without benefit. Examination showed small buried tonsils, cryptic and with a pustule on the surface of the right tonsil. I gave her a hand mirror and, without cocainizing, showed her the pustule, and with a paracentesis knife evacuated the same, explaining that very probably both tonsils were filled with these small pockets of infection, which could very reasonably account for her arthritis. This minor surgical procedure was done at her first visit at 12 noon. The patient was to have given a dinner party that night, of which she did not acquaint me until much later, otherwise I would have refrained from performing even such a minor operation.

Within an hour after leaving my office she began to feel a sensation of fullness and choking in her throat. By evening

she was aware of an increasing discomfort and difficult breathing. By 3 o'clock the symptoms were alarming to the patient, and she insisted I should leave the hospital, where I was operating, and see her at my office. I conjectured that it was only nervousness on her part which would not be substantiated by her complaints as anything serious.

At 4 o'clock, however, I saw her at my office, and a most alarming condition presented itself, in fact, so distracting that I asked the patient to excuse me for a moment and left the office so that I could concentrate upon what could be done for her, and what means could be adopted for her immediate relief. Examination showed the right tonsil and the whole of the lateral pharyngeal wall to be bluish red, edematous and infiltrated. The epiglottis was turban shaped and edematous, as was also the right arytenoid and aryepiglottic fold. The edema was so extensive that only a small breathing space was left, and the patient was speechless, cyanotic and near collapse.

Under local anesthesia, I punctured the epiglottis and arytenoid, evacuating a small amount of serum. I sent my assistant home with the patient, with the instruction to get a nurse at once, pack her neck in ice, give her crushed ice to suck, start a steam inhalation of oil of pine and benzoin under a croup tent, and to spray the throat frequently with alumnol, 20 grains to the ounce. He was directed to remain by her side until I could arrive with a tracheotomy set ready to do an immediate tracheotomy, as it seemed that the rapid progress of the infiltration and edema would necessitate this procedure. Before she left the office I gave her a hypodermic injection of a quarter of a grain of morphin and five minims of adrenalin. I also told my assistant to give repeated doses of Epsom salts until marked catharsis was induced.

As soon as I could prepare my instruments I went over to see her and found the edema lessened, but the epiglottis and arytenoid markedly inflamed, infiltrated and dusky red.

My assistant remained all night administering to the patient as previously instructed. At 7 the next morning the temperature was 100, pulse 74 and respiration 24. The edema of the larynx was markedly less. The treatment was continued with slight additional constitutional care, as colonic irrigations, etc., and in a few days all symptoms had disappeared except

an exceptionally large tonsil, which was very red, boggy, infiltrated and extending down to the epiglottis.

I had taken a culture in the beginning of this attack, which was reported as a pure streptococcus hemolyticus. As soon as an autogenous vaccine was available I administered it, but without apparent benefit. Her urine also showed a trace of albumin, with hyalin and granular casts, all of which ultimately disappeared.

There was an interim of quiescence, during which she was treated, but in May she developed a second infection which resulted in a typical peritonsillar abscess on the same side. This condition naturally created consternation in the mind of the patient, but immediate evacuation followed by treatment upon the lines of her first attack caused this trouble to abate without serious consequences. On June 12th, with many misgivings, I removed the tonsils under local anesthesia, but my anxiety was unwarranted, as the patient experienced no unusual difficulties and made an uneventful recovery. Since then she has markedly improved in every way, and has had no further throat trouble.

An unusual coincidence attaches itself to the history of the next patient, No. 3, which, from its similarity to the one just related, aroused in me the fear that misfortunes run in threes.

On the same evening of my trying experience with Mrs. G., at 9 o'clock I was requested by Dr. George Davis to see a woman in the Polyclinic Hospital, whose history was given to me over the phone as follows:

Mrs. W., fleshy, thick necked, healthy, but bereaved over the recent loss of her husband, had awakened early in the morning with a sticking pain in one tonsil and a sense of swelling or fullness in her neck and throat. By 10 o'clock the left side of her neck was distinctly swollen externally, while her left tonsillar region, uvula and palate were edematous. Her general physician, Dr. Burke, was called in, who verified these findings with the addition of an edematous epiglottis. He made an incision into the most pendulous part of the edema in the palate on the left side, and endeavored to obtain the services of Dr. Davis, a throat specialist, to see her immediately. Being unsuccessful, he got a general surgeon to see her in the afternoon, by which time she was quite dyspneic.



The general surgeon did not wish to assume responsibility for the case and only applied ice externally, preferring to await Dr. Davis' call, which was some hours later. Dr. Davis made a number of punctures in the uvula, epiglottis and left arytenoid.

She was then sent to the Polyclinic Hospital, where preparations were arranged for a tracheotomy. The next morning the symptoms had somewhat abated, and there was much less edema on the left side, but the right side now began to show a similar involvement. Gradually the symptoms progressed until there was marked evidence of exhaustion and the patient had great difficulty in breathing, besides becoming cyanotic. At 9 p. m., Dr. Davis called me to see the case and, after examination, I predicted that the patient would not live the night through unless an immediate tracheotomy was performed. In this Dr. Davis acquiesced, and I did a tracheotomy under local anesthesia, much to the immediate delight and comfort of the patient. The tube was removed on the fourth day, and she made an uneventful recovery. This case has been reported as an acute angioneurotic edema, but I disagree with this diagnosis, as it had all the earmarks of an acute streptococcic hemolytic infection.

Case 4.—Mrs. C. R. H. was referred to me in May, 1917, by Dr. Robert Reese for an acute catarrhal otitis media in the left ear. I treated her for various ear, nose and throat troubles at infrequent intervals until December 22, 1923. She was now 45 years of age, with no family or past history bearing on her present condition other than being somewhat fragile. She was taken suddenly ill with aches and pains throughout the body and a general feeling of fullness and irritation in her throat. She called in her family physician, Dr. H. C. Cowles, who found her temperature to be 102, her tonsils and pharynx hyperemic and a small patch of grayish exudate on the left tonsil. He at once had a culture examined for diphtheria, which was negative. He gave her a gargle and treated the case as one of ordinary follicular tonsillitis. On the following day the exudate had extended, the entire throat was markedly inflamed, the palate and uvula were edematous, and the glands of the neck on the left side enlarged and tender. On the third day, he called me to see the case in consulta-

tion. I found an extensive exudate covering both tonsils. The left supratonsillar region bulging as though a quinsy had started, and the exudate extending not only over the tonsils, but upon the palate as well. The distinctly grayish appearance of the membrane, the bleeding following the removal of the membrane, and the glandular involvement led me to strongly suspect diphtheria.

I took another culture from beneath the membrane, together with some of the membrane, to the laboratory, and advised the administration of antitoxin, which was done. The only organism found, however, was streptococcus hemolyticus. The temperature now was 105. Both sides of the neck were swollen and the glands enlarged and tender. The patient showed marked septic exhaustion, difficult breathing and edema extending down into the laryngopharynx. The method of treatment was hot irrigations of the throat every hour, followed by a gargle and an ice pack externally, with internal medication for her irregular and feeble heart action.

On the fourth day the condition was more exaggerated, and there appeared a pleuropneumonia at the base of the left lung. On the fifth day I was again called to see her, at which time she was too exhausted for me to make an accurate examination of her throat, but what could be seen gave evidence of advancement of the infection with some breaking down of the left tonsil. Her temperature had remained between 104 and 105, and there was now a pneumonia of the right lung. Her respiration varied between 40 and 60, and the patient gave evidence of extreme sepsis.

The next day, the 6th, the pneumonic processes in both lungs had extended so that the physician in charge said that there was no hope. She was by this time irrational and early in the morning of the seventh day she died.

Whether she would have died if pneumonia had not complicated the case is conjectural, but from the time I first saw her I was dubious of the outcome and so expressed myself to her physician and husband.

For verification of the etiology as expressed in this article, I submitted the paper to Dr. Jonathan Wright, who has kindly supplied me with the following history, resembling some of

those cited, and, with the permission of the president, I beg leave to read the same.

"Many years ago, on my return, somewhat shaken, from the festivities which used to grace the meetings of the Laryngological Association in Washington before the adoption of the eighteenth amendment, I found a hurried call awaiting me to a patient who is now Judge Blank. He was even then prominent both in his professional activities and in social life. His pharynx presented the conditions you have described so well in your cases, not only the extreme edema but festoons of serous blebs hung from the soft palate and uvula, and his epiglottis was edematous. His systemic condition was not so profoundly emphasized as you describe, but he was rapidly drifting into that state. For further horrors, however, he was a hemophiliac, whose external carotid had been tied by Sands after a tonsillotomy. In desperation, and to the terror of his mother, I plunged a bistoury into a prominence—a little pus and much serum—and blood, blood, blood for twenty-four hours. I called a surgeon. He had no external carotid to tie, remember. I think perhaps the surgeon proposed to put a ligature around the neck. I wouldn't swear to that, but something like it, I think. At my last gasp, not the patient's, I injected something through a syringe into the opening I had made into the little cavity.

"After thirty years, Judge Blank is still alive, but I have never been the same man."

44 WEST 49TH STREET.

## LXXXIII.

### COMMON COLDS.\*

BY SECORD H. LARGE, M. D.,

CLEVELAND.

There seems to be two types of colds—those spread by direct infection from others—the ordinary “droplet” type of infection, disseminated by coughing, sneezing, kissing, etc., and those caused by a lowered resistance enabling the bacteria or virus present to set up an active inflammation in the upper air passages. There can be no doubt that colds are contagious when we see a whole family suffering from colds at the same time, or note how colds spread through a whole school. On the other hand, one also sees small infants with colds, who have never been exposed to anyone having a cold. The transmission details are somewhat obscure. Certain people apparently are immune to colds, while others appear highly susceptible. It is not known whether a cold confers a transient immunity or not. If it does, the immunity is very short.

Colds are ushered in with the sensation of nasal stuffiness, sneezing, lacrimation and languor. In the first stage of a cold there is marked dryness in the nose, followed shortly by a congestion, with a resulting difficulty in breathing through the nose. This stage is followed by one of watery exudate or discharge which in turn becomes mucopurulent or purulent in character.

The theories as to the cause or origin of colds have been legion. Until the bacteriologic era was ushered in, exposure to cold and changes in temperature were given as the chief causes. Since then many different bacteria have been credited with the production of colds. Of these, the pneumococcus, streptococcus, staphylococcus, bacillus influenza, micrococcus catarrhalis and Friedlander's bacillus have been cited as the most frequently predominating organisms. Miss Tunni-

---

\*Read before the Section on Instruction of the American Academy of Ophthalmology and Oto-Laryngology, October, 1923.

cliff has discovered a small anerobic organism with which she has been able experimentally to produce fairly typical attacks in dogs. Some investigators claim that in one epidemic the pneumococcus is the prevailing organism, in another it is the streptococcus, while in still another it is the microccus catarrhalis. Gordon investigated the role played by the latter in the production of colds. He examined 110 people, none of whom had either been knowingly exposed to colds or had suffered from colds within a recent date. Cultures were taken from these persons in practically all months of the year, and yet there was no seasonal variation in the percentage incidence of the micrococcus catarrhalis. Of the 110 normal healthy persons 51, or 46 per cent, showed the presence of the micrococcus catarrhalis in the nose, nasopharynx or throat. He then studied 119 persons suffering from colds. These were observed from 24 to 48 hours after the initial onset of the symptoms. The micrococcus was found in 54, or 45 per cent, of these 119 people. So the organism was encountered in cultures from persons not having colds as frequently as from those having colds, the percentage incidence being 46 and 45, respectively.

Almost all investigators and nose and throat textbook authors are inclined to give credence to the idea that chilling of the body surface or some form of exposure to cold, either without or after overheating, has a decided factor in lowering the body resistance and allowing the bacteria or virus which may be present to set up an active inflammatory process in the upper respiratory tract. It has been shown that chilling of the body surface of animals causes a congestion of many internal organs. But Mudd, Grant and Goldman, in a series of experiments, have brought out the fact that chilling of the body surface does not cause a congestion of the mucous membrane of the upper air passages, as is the rather common opinion, but on the contrary produces a vasoconstriction with a resulting ischemia. They also noted a lowering of the temperature of the nasal and pharyngeal mucous membrane amounting in some cases to six degrees centigrade. They feel that this lowering of the temperature and the vasoconstriction produced so lowers the local resistance as to allow infection to occur.

Foster, in confirming some earlier observations by Kruse, prepared filtrates from the nasal secretions of individuals having colds. The nasal secretion in each case was blown into a sterile Petri dish and was mixed with 10 cc. of 0.8 per cent salt solution. This mixture was then well shaken up and passed through a small Berkfeld filter. Ten soldiers were then inoculated with this filtrate by placing three to six drops of it well back into each nostril. Nine of the men developed typical symptoms of a "cold" in from eight to thirty hours. These symptoms persisted for three to six days. Cultures and subcultures of the filtrate were made. Subcultures which had been incubated seven days were used. Filtrates from these subcultures were diluted with salt solution until the dilution of the original nasal secretion had reached a dilution of 1:90,000. Eleven soldiers were then inoculated with this diluted filtrate in the same manner as in the previous experiment with the ten soldiers. In from eight to forty-eight hours all of the eleven men developed acute colds. No definite microorganisms could be grown from any of the cultures of the filtrates. Under the dark field microscope a profusion of minute bodies were seen, but no definite morphology could be made out. Likewise with stained smears no bodies could be accurately determined as microorganisms. Foster feels, however, that because of the production of all the symptoms of a cold with filtrates of such high dilution from nasal secretions and because of certain changes, ordinarily interpreted as indicative of bacterial growth noted in the cultures and subcultures, which were not present in the controls, he is dealing with a virus which is the cause of common colds.

Still other investigators think that colds are not due to one type of bacteria alone but to one or more of a group of bacteria. Others claim that the bacteria usually found are not the cause of the cold but merely are present as the cause of a secondary infection superimposed on the unknown organism or whatever it may be that is causing the cold.

From the literature sent out by the various pharmaceutical houses, one would be led to believe that the cure of a cold depends only on the injection of stock vaccines of pneumococcus, streptococcus, bacillus influenzae, and the like.

Floyd has had very gratifying results with vaccines. Where the infection has been severe he isolated members of the streptococcus and pneumococcus groups and prepared vaccines. Early in the fall he gave three or four inoculations of the combination of these vaccines to each case. This was repeated in the spring. If a cold occurred in the interim the patient was given an inoculation during the first twelve hours of the onset. He reports that one or two injections brought about a rapid termination of the cold. In some cases he was unsuccessful, but in the majority the inoculations modified the severity of the colds usually endured and have also diminished their frequency. In some cases immunity from acute respiratory infections was secured, especially in those who were abnormally susceptible to infection, and in whom frequently one cold rapidly succeeded another.

Unfortunately, our results do not coincide with his. For a period of three years, in practically all cases of colds which came into our office, cultures of the nose, nasopharynx and the throat were made. The technic employed consisted of rubbing a sterilized platinum loop on the mucous membrane of the nose, nasopharynx and the throat. Culture tubes were then inoculated with these loops. In some cases the patient's own blood implanted on culture media was used. The nose and throat were then thoroughly washed out with sterile salt solution by means of a large metal syringe. Cultures were again taken with the platinum loop from the nose, nasopharynx and throat. Cultures taken before the washing showed many different kinds of bacteria, while in those taken after the washing no staphylococci or micrococci catarrhales were present but only streptococci and pneumococci. In persistent colds Friedlander's bacillus was often found. In some cases of tracheitis cultures were taken through the bronchoscope and Friedlander's bacillus, the streptococcus and the bacillus influenzae were those most frequently encountered. Vaccines were made in all cases having colds. When a patient returned having another cold, a second examination of the discharge from the nose, nasopharynx and throat was made. If the organisms were the same as were found on the previous examination, vaccine therapy was then instituted. Of all these cases only one was entirely free from colds for a period of two years, and only one returned for vac-

cine treatment when suffering from a cold. This patient believes he receives some benefit from the vaccine and that it hastens the termination of the cold. In no cases where autogenous vaccines were used were we able from an objective standpoint to notice any benefit—i. e., in the appearance of the nasal mucous membrane. In some cases having frequent colds, injections of vaccines were given as a prophylactic measure. These patients received two injections a week of increasing dosage for three weeks. Our experience would lead us to believe that these injections did not prevent the patients from contracting colds. In some cases of recurrent colds with the same bacterial flora, the patients thought that the vaccine was beneficial in the first attack, but in subsequent ones they felt it was useless. We have come to the conclusion that vaccines prepared from the usual pathogens, such as the streptococcus, pneumococcus, micrococcus catarrhalis, Friedlander's bacillus, etc., which were found in the nose and throat are practically useless either in the treatment of a common cold or as a prophylactic measure against colds.

Inasmuch as the exact cause of a cold has not been determined no specific treatment can be given. All of us have our favorite cold prescription, yet we feel that there is no drug or combination of drugs which will either abort or hasten the termination of a cold once established. Some men advise the giving of atropin or belladonna during the stage of watery exudation, while others say the best treatment for a cold is to aid local exudation, since it washes off the bacteria. Aspirin and argyrol have been the two most popular drugs employed in the treatment of colds by the laity. The taking of aspirin by a patient up and about is to be condemned because of its diaphoretic action, with the possibility of subsequent chilling of the body.

In our opinion the only treatment is: First, complete isolation of the patient in order to prevent infection of others; second, confining the patient to bed and keeping him at a uniform temperature to prevent chilling of the body surface with subsequent reinfection. A certain amount of comfort can be secured by using some of the well known local and systemic treatments.



To reiterate, we have found vaccines of practically no use in the prevention, or in the cure of common colds. Colds can be prevented to some extent by more close attention to hygiene—the avoidance of foul overheated air and of overcrowding, and by more strict regard for proper clothing. Many people are too heavily dressed, so that they perspire while in a warm room and then become chilled when they go out of doors. Special care should be exercised in keeping the feet warm and dry. By isolating patients with colds there would be no danger of direct infection from one person to another. Adenoids, obstructions to breathing, etc., should be taken care of surgically. In most cities there is a law compelling landlords to keep apartment houses at a temperature not lower than a certain minimum degree. There should also be a law, not only affecting apartment houses, but also theaters, concert halls, Pullman cars and the like, compelling those in charge to keep the temperature under a maximum degree and to secure sufficient ventilation. Much has been said about the taking of cold baths to harden one, but there are some persons who cannot be hardened, inasmuch as each time that they take a cold bath they catch a fresh cold. Cold sponging of the neck and chest seems to be of benefit to these people. Recently there have been many advocates of cold air baths, which seems to be quite logical.

There is so much work that is yet to be done on many such subjects as common colds that your council is hopeful that the day is not far distant when our Academy will be able to put into practice our great ambition—i. e., the establishment of a research laboratory in the interests of ophthalmology and otolaryngology.

LXXXIV.

AN UNUSUAL SYMPTOM OF MAXILLARY SINUS  
DISEASE.\*

BY GEORGE J. DENNIS, M. D.,

CHICAGO.

As a symptom of nasal or paranasal disease, cough is not unusual. As a symptom of maxillary sinus disease, cough is unusual unless it is admitted that it is not often recognized. As a rule the maxillary sinus is regarded as a comparatively silent area, and emphasis is laid upon the more obvious symptoms of pain, headache, tenderness and intranasal congestion.

In reviewing his records, the writer was impressed by the small number of cases in which cough as an outstanding factor in the symptomatology was mentioned, and also by the still smaller number in which this symptom was present unaccompanied by other local symptoms. This led to a somewhat extensive search of the literature, with the result that very little or no mention of cough as a valued symptom in the diagnosis of maxillary sinus disease could be found. From these personal records sixteen cases were selected in which cough was the predominant symptom, the other symptoms being either absent or occupying only a minor role. Four of the cases were without other symptoms, although a history of bronchial irritation was noted in each of them.

The first of these cases presented itself in the winter of 1918. The patient was a young woman of eighteen. She had been taken down ten days before with the usual symptoms of influenza, which was so diagnosed by her attending physician. During this period persistent cough was the most aggravating feature of the case. Fever was present, rising as high as 102.5°. Râles, both dry and moist, could be heard over the entire chest. On the tenth day an examination of the upper air passages was suggested and acceded to. Inspection of the pharynx, nasopharynx and larynx revealed only a general

\*Read before the Chicago Laryngological and Otological Society, April 7, 1924.

paleness with slight hyperemia over the arytenoids and of the vocal cords. The nose showed only a mild hyperemia of the membrane of the middle turbinates—no general turbinate congestion, no septal irregularity and no evidence of pus. Transillumination was negative for the frontal sinuses, but a marked shadow was present over the left maxillary region. Puncture was done and pus evacuated. The effect produced was singularly dramatic. Cough almost immediately ceased, the temperature dropped to normal within a few hours and all pulmonary symptoms had disappeared by the morning of the following day.

Bearing this case in mind, every patient with persistent or unexplained cough has since then been subjected to a careful search for maxillary sinus disease.

The second case of interest came in the winter of 1919. The complaint was that of continuous cough and an excessive amount of expectorated mucus during the preceding six weeks. The patient was a man of forty-five years. Examination disclosed a general pharyngitis, small submerged tonsils with infective crypts without inflammation, lingual tonsil somewhat enlarged and moderate congestion of larynx and cords; in the nose, a slight septal irregularity, marked increase in size of the left inferior turbinate, less hypertrophy of the left turbinate. No pus was visible. As the patient could neither spare the time nor money for a radiograph, he consented to immediate puncture of the left antrum. This was done and a considerable amount of pus was removed. At his request the opposite sinus was punctured with a similar result. Within twenty-four hours the cough ceased, and within two weeks the expectoration was reduced to a minimum. In this case no chest examination was made.

The third case was that of a physician of fifty-five years. Cough was persistent, accompanied by "choking spasms," to use his own term, lasting for hours until mucus in quantity was expectorated. This cough with the choking sensations had been present for two years. Besides other remedies, vaccines had been used during the previous seven months. Examination of the chest proved negative. There was a general pharyngitis, infection of the tonsils, especially the left, a lingual varix and a moderate congestion of larynx and cords. In the nose

there was a marked deviation to the left, boggy middle and inferior turbinates on the right side, nothing to be seen on the left on account of septal deflection. No pus was discovered. Transillumination showed a definite shadow over the right maxilla. Irrigation was difficult on account of the swelling of the membrane about the hiatus semilunaris. The cough and spasms ceased within the week and so far as is known have not recurred.

The fourth case was that of a man of fifty years, who came in the first month of 1923. He had been under treatment in various ways for a continuous cough, which began in the early summer of 1922. Associated with this cough were choking spells commencing about midnight and lasting until early morning. He speaks of them as spasms with loss of breath for short periods.

Examination: The pharynx somewhat dry and moderate congestion; the membrane of the postnasal space thickened and granular in appearance; the tonsils of medium size, submerged; a lingual varix; slight hyperemia of larynx and cords. The nose, a septum with little deflection but a small spur anteriorly on the left side; inferior and middle turbinates larger than normal. No pus visible. Transillumination, a shadow over both maxillæ. Puncture of both sinuses with abundant pus findings. Within the week the choking spells ceased. He was able to sleep uninterruptedly, and within six weeks the cough had decreased so that he was no longer annoyed by it.

A study of the other twelve cases disclosed the following points of interest:

1. Cough of a most persistent character dominating the picture.
2. The absence of positive findings by transillumination in eight of the cases.
3. The positive findings by radiogram in all of the cases against the doubtful findings by transillumination.
4. The presence of bronchial symptoms in most of them.
5. The low grade of pharyngeal and laryngeal irritation as compared with the amount of cough.
6. The presence of pus, easily demonstrable on the side of the affection, as distinguished from the first four cases.

7. The rapid diminution of the cough after the first removal of pus without any relation to the continuation of the secretion. Other phases of these cases were not out of the ordinary.

While such phenomena as we have exhibited have received little recognition except in the most casual way, one communication only being found in which a study of the relation of maxillary sinus disease was made (that of Reider Gording in 1920), there appeared to be no end of clinical reports, experimental studies and speculation about the connection between nasal disease and all sorts of affections as varied as migraine and dysmenorrhea. The major part of these writings were devoted to the demonstration of the reflex relationship between definite points of irritation of the nasal mucous membrane and asthma. Much of our knowledge of the reflex paths between the nose and the central nervous system, and between the central nervous system and the lungs and heart, has been the resultant.

The writer trusts that he may be pardoned if he quotes rather copiously from the work of Gording, whose resumé of the literature was exhaustive and whose own experiments were prompted by four disastrous experiences in the puncture of the maxillary sinus, together with several other such experiences on the part of his colleagues. He collected reports of nine other cases, making twenty in all, and among the twenty, four deaths occurred.

According to him, Voltolini was the first to recognize the connection between nasal and bronchial affections. Fränkel, however, first called attention to the intimate relationship between nasal polypi and spasm localized in the bronchial muscles. As early as 1870, Kratchmer experimented with curarized rabbits, employing various gases, among them tobacco smoke, in an effort to trace the connection between the irritation of the nasal mucosa and its effects on respiration and on circulation. He arrived at the conclusion that the relationship was a reflex one, the sensory paths of the reflex connection being situated in the trigeminus, the motor in those nerves supplying the respiratory muscles and the vagus, and that the nasal irritation manifests itself in an expiratory tetanus, with expiration cessation of respiration accompanied by closure of the glottis, slowing

of heart action and increasing blood pressure. He considered reflex irritations of the larynx of no great importance. Reflexes there can be started but act differently, the above symptoms coming from the nose and not from the organs below. The nasal reflexes were set up, notwithstanding intracranial section of the olfactory nerve, but did not appear with destruction of the trigeminus. He considered that he had at least proven the sensory portion of the reflex arc. Knoll's findings, whose work was done on unnarcotized animals, were much the same as Kratchmer's. He went further, however, maintaining the view that not only must a connection be assumed between the sensory paths and the respiratory center, but that there must be a direct connection between the sensory nerves and the spinal which supply the respiratory muscles with motor fibers. If this were not the case, it would be difficult to establish the fact that in an animal patient, in which spontaneous respiration ceased with section of the spinal cord, a single inspiration could be called forth by strong electric stimulation of the sensory nerves. Irritation of these sensory paths can presumably cause a direct irritation not only of the respiratory center itself but also of the spinal centers for the respiratory muscles. This interference of irritants issuing partly from the respiratory center, partly from the spinal centers, conceivably may explain the numerous combinations of increased frequency and depression of the diaphragm found in animal experiments where now the one and now the other type of respiration prevails. The respiratory center situated in the medulla oblongata, whose rhythmic action is kept up by the constant flow of blood to it, can, on the one hand be excited by psychic or sensory stimulants and on the other be inhibited through the influence of certain tracts—e. g., the vagus and the trigeminus. The impulses that issue from the respiratory center are transmitted to the respiratory center in the spinal cord. It is probable, however, that the respiratory center may also be exposed to irritation directly from the cerebrum, as the expiratory muscles can, as we know, be voluntarily set into activity.

Sandman, carrying on further investigations confirming Knoll's results, claimed that in addition to expiratory cessation of respiration the nasal mucous membrane irritation caused

a narrowing of the air passages by contraction of the bronchial muscles. In this way the protection of the respiratory organ is increased and at the same time the removal of mucus from the alveoli and bronchi facilitated. By contraction of the smooth muscular fibers, with which the entrance to the alveoli is surrounded, the mucus will be detached and thus more readily removed from the infundibulum.

This author asserts that by his experiments on animals he has supplied a physiologic basis for the nasal asthma so frequently described in clinical reports.

Brodie and Dickson also claim to have proved contraction of bronchial muscles after intranasal irritation.

The experiments of Lazanes amount to the same thing. He claims the centrifugal part of the reflex is to be sought in the vagus, as after section of this nerve no effect is obtained, while increase of pressure in the bronchi again occurs on peripheral irritation of the vagus.

Killian quotes Grossman's experiments, showing that electrical and mechanical stimuli, in artificially breathing animals, of the posterior turbinates caused considerable functional disturbance of the heart action and of respiration. There occurs stasis of the pulmonic circulation, increase of the intrathoracic pressure and depression of the diaphragm. The reflex arc is supposed to go from the sensory nasal nerves to the central nervous system and thence through the vagi to the heart and lungs and through the phrenic nerves to the respiratory muscles. Grossman thinks with Koblack and Roder that the reflex must have taken another direction, which Killian believes will be in the sympathetic system. Möllgard, in 1910, made minute investigations of the respiratory system of vertebrates and confirms the view held in accordance with physiologic research, that the vagus carries bronchomotor nerves to the lung, but at the same time points out that these are of sympathetic character, as they are interrupted in the ganglion nodosum.

These investigations were carried on for the purpose of emphasizing the reflex nature of asthma, to regard it as a neurosis originating in various organs, very frequently the nose, whether the centrifugal paths are to be found in the vagus, the phrenic or sympathetic nerves.

More recently Sluder has demonstrated by careful analysis of clinical cases, by experiments and by anatomic studies, that practically all sensory impulses originating in the nose find their way to the sphenopalatine ganglion, from which they radiate to the vagus, sympathetic and other portions of the trigeminus. With these you are doubtless familiar.

Returning now to the work of Gording, much of it has no special bearing on the subject in hand, yet being the first to prove experimentally the effect produced upon the pulmonary and circulatory system by the introduction of irritants into the maxillary sinus, his findings may be of interest. He first injected air into the sinus, carefully excluding the nasal pharyngeal and tracheal membranes from contact with the injected air. The injection was immediately followed by these respiratory disturbances: 1. Forced respiration, succeeded by one or more superficial respiratory movements; 2. The respiratory movements become slow and irregular; 3. Expiratory cessation of respiration ensues. This cessation of respiration, which may be prefaced or concluded by one or two convulsive respiratory movements, continues without interruption as long as the irritation operates, or it may be succeeded by small irregular respiratory movements. The injection of air has no serious consequences for the animal. If the injection is continued for a sufficiently long time the respiration begins again, notwithstanding the continued pressure. The pulse does not seem to be affected in any way. Herein his experiments with air injection differ from those of the previous experimenters. Most of them, by irritation of the nasal membrane, realized a marked vagus effect. On the contrary, in his experiments the pulse curve and the blood pressure remained the same. Using a more powerful stimulus, such as alcohol, an injection into the right maxillary sinus produced instantly a violent reaction. There was an absolute cessation of respiration for eight seconds, only interrupted by one convulsive inspiration and then complete respiratory tetanus with a long series of convulsive movements, which did not cease until thirty-five seconds had elapsed. Death was momentarily expected. Respirations, however, began again with as strong respiratory movement as before the application of the irritant. During



the respiratory paralysis there also occurred general paralysis with violent jerks in all the extremities and in the face. Of no less interest were the blood pressure and the pulse curve after the alcohol injection. Whereas the blood pressure had remained unchanged in the previous experiment, in this one we find a tremendous increase, which remained high throughout the respiratory phase, but showed a somewhat falling tendency, so that when respirations began once more the pressure was 24 mg. Hg. lower. The pulse curve, too, was changed constantly. The beats at first became slower, then small and irregular, and finally during the greatest rise in blood pressure seemed to disappear entirely. During the spasms the pulse curve appeared as an almost straight line; but as the convulsive stage was passed and the respirations began again, the pulse beats were seen once more in the curve, which acquired the same appearance as it had before the injection.

These experiments appear to prove that irritation of the maxillary sinus may result in much the same phenomena as in irritation of the nasal mucous membrane. It would be interesting to follow with Gording his conclusions as to the reflex paths which the nervous impulses in his opinion seem to follow, but this paper is already too lengthy.

The impressions produced by a study of these personal cases were that they must be of a reflex character. Conceivably they might be of toxic origin, yet the rapid subsidence of the symptoms after the removal of the contents of the sinus seemed to proclaim the reflex rather than the toxic nature of the symptoms.

LXXXV.

LEFT TEMPORAL LOBE ABSCESS WITH A REPORT  
OF TWO CASES.\*

By C. F. YERGER, M. D.,

CHICAGO.

The temporal lobe is the most frequent site of brain abscess. The combined statistics of many authors show that the great majority of brain abscesses are of otitic origin. Brain abscess is more frequently a complication of chronic than of acute otitic suppuration. Jansen,<sup>1</sup> in the Berlin Ohrenklinik, found one brain abscess in 2,650 acute and six in 2,500 cases of chronic suppurations. Dickie,<sup>2</sup> from the reports of the Royal Infirmary of Edinburgh, gives the frequency of brain abscess to otitic suppuration as four per thousand. Otogenous brain abscess may result after the middle ear suppuration has apparently been cured, but where the cavum tympanum and tegmen are diseased. Henius<sup>3</sup> reported twenty such cases, the longest interval between the cessation of the ear disease and the appearance of the brain abscess being eight months. The interval between the onset of the otitic infection and the development of a brain abscess is variable. Bezold<sup>4</sup> observed a case that began 46 days after the onset, and Schwartze<sup>1</sup> had a patient who developed a brain abscess after having a chronic suppurative ear disease for 44 years.

The site of otitic brain abscess is usually found adjacent to diseased bone. Koerner<sup>4</sup> collected 100 cases of brain abscesses of which 42 per cent had fistulous openings connected with diseased bone. Pachymeningitis externa and interna are generally associated with the diseased bone. The brain is not always adherent to the meninges when a brain abscess is present. A brain abscess may be present without having any visible connection with the diseased bone.

The frequency of temporal lobe abscess can be explained on anatomic grounds. The middle ear is separated from the

\*Read before the Chicago Laryngological and Otological Society, April 7, 1924.

cranial cavity by the tegmen tympani and antri, one of the thinnest parts of the floor of the cranial fossa. The petrosquamosal suture may contain blood vessels which connect the tympanic with the cranial cavity, or it may be the site of a dehiscence. Abscess of the temporal lobe usually occupies the inferior temporal convolution, and Koerner<sup>4</sup> states that the abscess is usually located a few mm. within the cortex.

The symptoms of brain abscess may be conveniently divided, according to von Bergmann, into three groups: First, those due to the presence of infection, as fever, leucocytosis; second, those due to increased intracranial pressure, as slow pulse, optic neuritis, and third, the focal symptoms, which are of value, both in the diagnosis and in the localization of the brain abscess. It is, therefore, the latter group that we shall consider more or less in detail.

A large brain abscess may be present in the right temporal lobe without causing any focal signs, while a small abscess in the left temporal lobe may cause very characteristic aphasic disturbances. Aphasia rarely occurs in an abscess of the right temporal lobe. Oppenheim saw but one case. This is readily explained, when we recall that the sensory cortical centers and the association tracts that have to do with an aphasia are located in the left temporal lobe in right handed individuals. The cortical center for motor speech is located in the left inferior frontal convolution (Broca), a lesion of which produces motor aphasia, the patient losing the power of speech. Koerner<sup>4</sup> states that no motor aphasia has been reported in uncomplicated cases, and Macewan<sup>5</sup> never saw a case of temporal lobe abscess with motor aphasia. In the left temporal lobe is located the auditory word center, the center for understanding speech or for the memories of words heard. It is located in the posterior two-thirds of the superior temporal convolution (Wernicke) and supramarginal gyrus (Marie), a lesion of which causes sensory aphasia (Wernicke) or word deafness (Kussmaul). The visual word or reading center is located in the posterior part of the left temporal lobe and angular gyrus, a lesion of which produces alexia, in which the printed or written word can be seen but they have no meaning. Alexia is frequently associated with hemianopsia,

while optical aphasia, amnesic aphasia and paraphasia are usually associated with word deafness.

In 1889 Freund<sup>6</sup> described a condition which he named optical aphasia, which is pathognomonic of left temporal abscess. It is characterized by an inability of the patient to give the name of an ordinary object which he sees and recognizes. However, the patient can tell for what the object is used or how it is used, and, with the aid of the other senses, as taste, touch, smell and hearing, the patient may be able to name the object. The instant the patient hears the name pronounced he will recognize it and will be able to pronounce it. The optical aphasia may be incomplete—i. e., the patient may be unable to name some common objects, but is able to name others. The lesion, according to Freund,<sup>6</sup> is located in the left temporal lobe and involves the association tracts between the visual center in the occipital lobe and the speech center. According to Oppenheim,<sup>7</sup> the abscess is so situated that the tracts which connect the sound memory center with the optical center is disturbed and the probable localization is in the basal and posterior portion of the temporal lobe. Ballance<sup>8</sup> states that anomia, or the loss of the power of naming objects, is due to a paralysis of the naming center, which is located in the posterior part of the left inferior temporal convolution.

The literature is replete with clinical confirmation, both from operation and autopsy, that an abscess in the white matter of the posterior part of the left middle and inferior temporal convolutions causes optical aphasia. The importance of optical aphasia in the diagnosis of left temporal lobe abscess cannot be overestimated; it not only aids in the diagnosis but localizes the abscess. In the cases herewith reported, the diagnosis of brain abscess and the decision to operate was made practically upon this finding. The statement that Oppenheim<sup>7</sup> makes, that optical aphasia is often overlooked or is unrecognized by otologists in many cases of brain abscess, no doubt is true. Sensory aphasia was the guide to the diagnosis in the first successful operation in otitic brain abscess (Schede). This opened up a new era in brain surgery; since this many cases have been saved by an early diagnosis and treatment, likewise many cases have succumbed for the lack of it.

Up to 1899, Blau<sup>9</sup> states that 83 cases of left sided otitic temporal abscesses were reported, of which 50 had disturbances of speech. In 12 cases there were no descriptions of the kind of aphasia present. Word deafness, pure or mixed, was present 7 times; paraphasia, 11 times; amnesic aphasia, 22 times; alexia and agraphia, 4 times; optical aphasia, 3 times; and motor aphasia, 3 times. Schmiegelow<sup>4</sup> collected 54 cases from the literature of left temporal lobe abscess, of which 23, or 42 per cent, had speech disturbances—i. e., used wrong words (paraphasia) or were unable to name objects (optical aphasia). Koerner<sup>1</sup> states that in otitic temporal lobe abscess there is frequently a lesion of the internal capsule resulting in contralateral phenomena, viz., paresis, rarely paralysis, clonic or tonic spasms, hemianesthesia and homonymous hemianopsia. Mygind,<sup>10</sup> in 43 cases of abscess of the temporal lobe noted paralysis of the opposite side in 7 cases, paresis in 3 cases and ptosis in 4 cases.

The early symptoms of a lesion of the left temporal lobe are amnesic aphasia or the inability to remember names, and optical aphasia, or the inability to name objects; the late symptoms are word deafness, word blindness, right homonymous hemianopsia, right hemiplegia and right hemianesthesia.

The probability of the presence of an homonymous hemi- or quadrant anopsia in temporal lobe abscess emphasizes the necessity of taking the visual fields in all suspected cases. Cushing called attention to this when he reported a large number of temporal tumors in which he found quadrantic anopsia. Pollock,<sup>11</sup> found this associated with an aphasia in a case of left temporal abscess.

Optic neuritis or choked disc, according to Ruttin and Neumann, occur far more frequently with cerebellar than with temporal lobe abscess. Statistics of various authors show that less than 50 per cent of brain abscesses show optic nerve changes. Fortunately, according to Macewen, the optic neuritis is rarely followed by atrophy and the sight generally returns when the intracranial pressure is relieved. Delayed cerebation is characteristic of temporal lobe abscess. Slow pulse is a very important diagnostic sign and according to Macewen may be the sole indication for operation in some cases.

Probably the most important procedure in the diagnosis of a suspect suppurative intracranial lesion is the examination of the cerebrospinal fluid obtained by lumbar puncture. In the very early stage of brain abscess the spinal fluid shows slight but significant changes; the spinal fluid is under increased pressure, is clear or slightly turbid, depending upon the amount of cells present, which varies from slightly above normal to usually less than 100 cells, in which the lymphocytes predominate, globulin is increased and the smear and culture are negative for microorganisms. Brain abscess is associated with a protective<sup>12</sup> or sympathetic meningitis;<sup>13</sup> the picture of the cell count varies with the stage of meningeal irritation present. As the case progresses in intensity, the tendency is for the cell count to increase in size and to change from a lymphocytic to a polynuclear predominance. In 17 cases of brain abscess observed at Cook County Hospital, in which the diagnosis was confirmed either by operation or autopsy, the cell count was under 400 in 65 per cent. If, during the course, a sudden and marked increase in the cell count is observed, especially if it is over 10,000, it should suggest the presence of a complicating septic meningitis. As a rule, a very high cell count speaks for septic meningitis but of this we cannot be certain unless we find the microorganisms in smear or culture. Boot,<sup>14</sup> reports two cases of brain abscess which recovered that had high cell counts, one with 6,250 and one with 16,300 cells. These were cases of sympathetic meningitis.<sup>15</sup>

Among the 17 cases above mentioned, the minimum cell count was 8, which is normal if they were lymphocytes and abnormal if they were polymorphonuclear cells; the maximum cell count was 44,400 cells; both cases showing no meningitis at autopsy. Only three of the 17 cases had more than one lumbar puncture, less than 2 per cent. Parenthetically, I wish to emphasize the necessity of more frequent examination of the spinal fluid for the proper study of these cases. Nine of the thirteen cases that were autopsied, or 70 per cent, showed meningitis. In one case, (B. A. 1920), the cell count was 60; the abscess was drained; with the development of a fatal meningitis the cell count rose to 56,000. The case herewith reported, (B. A.), is analogous; in the sympathetic

meningitis stage, the cell count was 94; two months after operation, when she developed a fatal meningitis, the cell count arose to 21,400 and the culture showed streptococci. Septic meningitis is the most frequent terminal complication of brain abscess and when it occurs there is usually a sudden and a tremendous increase in the cell count together with the finding of bacteria in the culture.

The treatment of brain abscess is operative as soon as the diagnosis can be made. A temporal lobe abscess is invariably of otitic origin and therefore the ideal operation is thru the avenue of infection—i. e., mastoid and tympanic cavity, for the following reasons: because this route is less dangerous for the patient; 2, nature has in the great majority of cases already cofferdammed this approach to the brain abscess by sealing off the subarachnoid space; 3, because this route gives local evidences of the location of the brain abscess in the majority of cases, viz., the reddish area of pachymeningitis, granulations on the dura, the fistulous connection with diseased bone, and localized bulging with lack of pulsation; 4, the site of the brain abscess is most often found near diseased bone; 5, the most dependent route, gives the best drainage on account of gravity; 6, because the associated and causative infection should also be cleaned up and this necessitates a radical mastoid operation.

Brain surgery owes its gravity to the existence of the subarachnoid space; if it were not for this, the drainage of a brain abscess would not be such a formidable surgical procedure. Where there is a spontaneous walling off or exclusion of the subarachnoid space by nature, forming protective adhesions, this danger becomes practically negligible; but, in the absence of this, what is the best method of procedure? Either one of two procedures can be adopted: 1, to disregard the absence of a spontaneous exclusion of the subarachnoid space; 2, to induce walling off or exclusion of the subarachnoid space by producing protective adhesions. Lemaitre,<sup>15</sup> accomplishes this by the use of a filiform drain, gradually increasing the size of the filiform until adequate drainage has been established. Ballance,<sup>8</sup> does a two stage operation; he exposes the dura and 24 to 48 hours later, after adhesions have formed between the dura and piaarachnoid, he drains the abscess. Boenninghaus,<sup>16</sup> claims that 50 per cent better

results can be obtained with his technic which consists essentially in the formation of a tubular shaft to correspond to the diameter of the opening into the skull which extends into the brain abscess cavity. He attains this by the removal of the brain substance with a conchotome, alongside of the puncture needle; packing the abscess cavity and shaft thus made with gauze which is later replaced by a drainage tube. The advantages he claims for this method are that it avoids contusion of the brain and the formation of a dead space and that meningitis and encephalitis are prevented. Starr,<sup>17</sup> has seen brain abscesses evacuated through too small an opening subsequently develop fatal meningitis. The majority of authors are in favor of a small opening in the dura; the advantages of which are less liability of infection of the subarachnoid space and the prevention of brain fungus and hernia.

An opening of the dura of sufficient size for the drainage can be made with a Gifford brain abscess forceps by puncturing the dura and then spreading the blades of the forceps sufficiently to admit a  $\frac{1}{4}$  or  $\frac{1}{2}$  inch rubber drainage tube into the brain abscess cavity. As the most important part of the after treatment is to provide adequate and continuous drainage, the drainage tube should not be removed permanently until all discharge has ceased—i. e., before three or four weeks; but the tube should be removed daily for cleansing purposes. I think it is a mistake to substitute gauze for the drainage tube, as this favors premature constriction and closure of the dural opening. If the opening in the dura has been constricted too small for drainage, it should be enlarged by spreading the blades of a forceps and dilating the constriction. As the tendency is toward a premature closure of the operative wounds before the necessity for drainage has passed, all exuberant granulations must be removed with stick nitrate of silver or scissors.

The following is the report of two cases of left temporal abscess observed at Cook County Hospital.

Case 1.—R. C., age 9, became ill with scarlet fever, 19 days ago (June 16, 1923), with spontaneous rupture of both drum membranes five or six days after the onset; both ears have been discharging since. A right facial paralysis was noted soon after rupture of the drums.



Physical examination on admittance: Temperature 101.4, pulse 120, respiration 24. A noticeable deafness, bilateral otorrhea and a fluctuant, red, tender swelling over the left mastoid are present. She is unable to close the right eyelid and the right corner of the mouth does not move when the mouth is opened. White blood corpuscles 7,600.

Diagnosis: 1. Bilateral acute suppurative otitis media. 2. Left acute suppurative mastoiditis with subperiosteal abscess. 3. Right Bell's palsy.

Operation, June 22, 1923. Simple mastoidectomy (left). Large subperiosteal abscess drained; no fistula found; mastoid cells contained pus; mastoid tip necrotic; small subdural exposure.

Postoperative course: The temperature arose to 102.4 and on the second day to 104; white blood corpuscles, 12,500; on the third day the spinal fluid was under pressure, globulin increased and contained 240 cells, mostly P. M. N., and on culture the fluid was sterile. A moderate cervical rigidity was present, Kernig, Brudzinski, Babinski and Oppenheim were positive. The hearing was greatly diminished in both ears but it was impossible to determine this quantitatively because of lack of cooperation. The right ear was irrigated with cold water at 68° for four minutes without producing a nystagmus.

The spinal fluid and the meningitic signs pointed to a sympathetic meningitis due to some meningeal irritation, the labyrinth test to a dead labyrinth or at least to involvement of the labyrinth. Accordingly, it was decided to explore the previously operated or left mastoid and the middle cerebral fossa for extradural, subdural or cerebral abscess and to explore the right mastoid on account of involvement of the facial nerve and labyrinth.

Operation, June 26, 1923. Through the kindness of Dr. Alfred Lewy, who placed at my disposal some vucin for use in meningitis cases, an intraspinal injection of 10 cc. of a 1:750 solution of vucin was made after withdrawing 20 cc. of spinal fluid, which was clear, under pressure, globulin increased and contained 37 cells. A left radical mastoid operation with exploration of middle cranial fossa and temporal lobe. No pus or bony necrosis found; no pachymenin-

gitis; the dura did not pulsate. After removal of the tegmen tympani and antri, the dura was punctured with Gifford's brain searcher and the temporal lobe explored for brain abscess, going to a depth of 2 cm. in an upward direction, anteriorly and posteriorly without finding any pus. Between the blades of the brain searcher a strip of iodoform gauze was inserted through the opening in the dura and into the brain cortex with the idea of cofferdamming the subarachnoid space. The tegmental region, cavum tympani and mastoid cavity were lightly packed with gauze. Right radical mastoid with exposure of middle cranial fossa. The pneumatic cells contained pus but the walls had not yet become softened; the dura was exposed in the tegmental region and was of normal color; but it did not pulsate; no exploration of the brain or labyrinth operation was done.

Postoperative course: A spinal puncture was done the next day which showed pressure increased, cloudy, globulin increased and cultures sterile. Another intraspinal injection of 10 cc. of 1:750 solution of vucin was given. A marked improvement was noted two days after the operation, this was especially noticeable in her mental condition. The Kernig and Brudzinski disappeared gradually; on the fifth day she became afebrile; on the ninth day she commenced to run a slight fever; on the eleventh day she complained of severe headaches, and there was a slight rigidity of the neck, temperature 101, pulse 128, respiration 28; on the twelfth day she became apathetic and stuporous but could be easily aroused and would answer questions intelligently, temperature 101.4 to 103.4 and pulse 128 to 154, neck rigidity, Kernig and Brudzinski positive. Spinal fluid pressure diminished, cell count 650, 73 per cent P. M. N., globulin increased and sterile. Optical aphasia present as she was unable to give the name of common objects as keys, safety pin, knife and fountain pen shown her.

Diagnosis: Left temporal lobe abscess and sympathetic meningitis.

Operation: The left mastoid wound was opened and the tegmental region exposed; a pachymeningitis externa was present; the dura was punctured with the brain searcher and introduced into the temporal lobe, upward and anteriorly for

1.5 cm., which drained a brain abscess from which 8 cc. of pus was evacuated; a  $\frac{1}{4}$ -inch rubber drainage tube was inserted into the abscess cavity.

Postoperative course: Within 48 hours the patient was greatly improved; the fever disappeared and has since remained practically normal. The optical aphasia was present on the fourth and fifth days, but later disappeared completely. On the nineteenth day she was able to read and write fluently and there were no signs of optical aphasia. Considerable difficulty was experienced in keeping down granulations and keeping the wound from closing too soon. The drainage tube was dispensed with after thirty days.

Comment: The Bell's palsy was of toxic origin coming on within a few days of the onset of the acute otitis media. A left mastoidectomy with drainage of a subperiosteal abscess was performed 22 days after onset after which she did not improve but became worse. Four days later when she developed signs of a sympathetic meningitis, a left radical mastoid operation with exploration of the middle cranial fossa and temporal lobe; also, a right radical mastoid operation with exposure of the right cranial fossa were performed. Following this, there was a period of marked improvement for nine days, after which she again developed symptoms and signs of a sympathetic meningitis and on the twelfth day after the temporal lobe exploration, or 38 days from the onset, optical aphasia appeared and a left temporal abscess was diagnosed and drained, after which she made an uneventful recovery. Query: Was the left temporal lobe abscess present at the time of the left temporal lobe exploration or did it result from this procedure? If the latter was the case, how can we account for a cell count of the spinal fluid of 240 and 37 cells before the exploration, and would not a right simple mastoidectomy have been sufficient interference?

Case 2.—B. A., age 24. Onset of acute otitis media about three weeks ago; left paracentesis was done, since which her left ear has been discharging. About a week later she became so acutely ill she had to go to bed; she had attacks of vomiting usually after eating or drinking; complained of severe headaches and suffered from nausea. She states that she never had any previous trouble with left ear, but had a right mastoid

operation a year ago. She now complains of pains over the entire head and has attacks of vomiting without nausea. Physical examination reveals a well nourished young woman, apparently very sick. Temperature 98.4, pulse 52, respiration 20. The left auditory meatus contains purulent material; there is a perforation in the posterior quadrant of the left drum membrane from which a small amount of seropurulent fluid exudes, and slight mastoid tenderness is present.

The fundus examination shows a bilateral papilledema. An optical aphasia is present, i. e., there is a loss of memory for names of various common objects. The spinal fluid was under pressure, clear, cell count 94, globulin increased, Wasserman 3+.

Diagnosis: On account of the presence of A. O. M. S. with probable mastoiditis, together with the presence of signs and symptoms of intracranial pressure, (vomiting, headache, papillitis, slow pulse, increased spinal fluid pressure and cell count), in the absence of meningitic signs, (stiff neck, Kernig, pathologic reflex signs, and a low cell count), and the presence of an optical aphasia in a right handed individual, the trouble was localized as an abscess in the left temporal lobe.

Operation, May 19, 1923. Left radical mastoid operation and exploration of the left temporal lobe with drainage of temporal abscess. The mastoid was diploetic and free from pus, no large pneumatic cell found except the antrum; the cavum tympani contained cholesteatomatous material, granulations and pus. The tegmen was covered with granulations; it was removed and the supratragmental region of the dura was exposed; the dura was red and granulating and thickened, i. e., a pachymeningitis externa was present. The brain did not pulsate. Without making an incision into the dura, Gifford's brain searcher was inserted through the dura and into the temporal lobe, for a distance of 3 cm., which liberated about 60 cc. of a very fetid, thick, purulent fluid mixed with cheesy flocculi. A nonperforated  $\frac{1}{4}$ -inch rubber drainage tube was inserted into the abscess cavity. The supratragmental, tragus, and mastoid regions were packed with iodoform gauze. Culture from the brain abscess pus showed streptococci and colon bacilli.

Postoperative course—Comment: The postoperative course was uneventful, until the sudden appearance of her fatal illness, a septic meningitis, which caused her death, July 20th, 62 days after the operation. As a rule, the abscess drained profusely, until the day before she died. The drainage tube was removed ten days after the operation and gauze substituted. This was a mistake; first, because the gauze did not act as a drain, but more of a plug, as 17 days after the tube had been removed when the abscess was not draining well, the abscess cavity was probed and 15 cc. of pus escaped; second, the gauze is not so readily inserted into the abscess cavity as the tube; and third, the neck of the abscess will close up more readily over gauze than a rubber tube. In spite of the fact that there was great difficulty in keeping the wound from closing up on account of the natural healing tendency, together with the persistent presence of exuberant granulations, the drainage was very profuse. It seemed almost incredible that such a large quantity of pus could be discharged through an opening not sufficiently large enough to admit an infant's catheter. A probe inserted into the abscess cavity showed that it had not been reduced materially in size since the operation. The fact that the discharge in such quantity kept up so long became grounds for fear and apprehension lest the prognosis might not be so favorable as was anticipated, but in view of the fact that the drainage was so good, it was thought best not to take any chances on enlarging the dural opening for fear of causing a rupture of the adhesions and opening up and infecting the subarachnoid space, causing a septic meningitis. In view of the fatal outcome, caused by a septic meningitis, without the contemplated operative interference, this was undoubtedly a mistake. The brain abscess continued to drain until the day before she died; the day she died the abscess cavity was explored but no pus was found. The autopsy showed a very slight amount of pus in the abscess cavity in the temporal lobe; no rupture could be demonstrated into the subarachnoid space or lateral ventricle.

The temperature and pulse record shows that the temperature was often normal and rarely reached 99; 58 days after the operation and 4 days before she died, the temperature reached 101 after a chill. The pulse in the first four days

following the operation ranged from 60 to 84 and later on became faster up to 100.

In view of the fact that the localization of the brain abscess was made solely on the sensory aphasia present, it will be worth while to examine a little more closely into this phase of our patient's symptomatology. Before the operation, the patient had optical aphasia—i. e., a loss of memory for names of some common objects, as keys, knife, etc.; two days after operation Dr. Krumholz found no evidence of a motor or sensory aphasia and her sensorium was in no way affected; 19 days after operation her mind seemed bright and active and her memory was keen and accurate for past events; 33 days after operation she had amnesic aphasia; she had great difficulty in remembering names and repeatedly forgot her aunt's name with whom she lived and who had been coming to see her at the hospital two or three times a week; she forgot her sister's name and could not remember the name of the city in which she was born and where her mother now lives in Denmark; 44 days after operation, another examination by Dr. Krumholz showed the presence of an optical aphasia; she could not recognize the names of objects and was conscious of this defect; she could read and write and could speak and write from dictation and she understood spoken language; 62 days after operation, on the day she died, optical aphasia was absent.

About a month after operation, she began to put on weight; she gained 10 to 15 pounds and was feeling fine and begged to be allowed to go home. She was given antiluetic treatment on account of a 3 + spinal fluid Wassermann.

During the last month of her illness, she complained of being dizzy at times, at which time she showed a tendency to fall towards the right. She never showed any spontaneous nystagmus or past pointing. These attacks of vertigo were in all probability due to involvement of the vertigo center in the posterior portion of the superior temporal convolution.

Her fatal illness, in all probability, began with a chill on July 16, on the 58th day since the operation and four days before her death. Her temperature had been normal in the morning but following a chill, it reached 101°. The abscess was draining well, although the opening in the dura had

become very small. On the two following days, the drainage was profuse and on the day before she died, the drainage stopped. She complained of headache, pains in head and neck, vomited, was very restless and her temperature ranged from normal to  $102.4^{\circ}$ . On the day she died she complained of feeling extremely tired and weak; she vomited profusely and frequently. She was stuporous but could be aroused and answered questions. She could recognize and give the correct name of common objects. The neck was slightly rigid, tache cerebrale was marked, slight left optic neuritis, and slight paresis of both internal recti were present. The spinal fluid did not show any increased pressure, but showed a very turbid fluid, a cell count of 21,400, 95 per cent P. M. N., globulin positive. Smear showed Gram positive cocci and culture showed streptococcus hemolyticus.

Diagnosis: Septic meningitis.

After withdrawing 20 cc. of spinal fluid, 15 cc. of a 1 to 750 solution of vucin in a 50 per cent normal salt solution was injected intraspinally.

Exploration of brain abscess July 20th, the day she died. Gifford's brain searcher was inserted through the former site of drainage into the abscess cavity without obtaining pus; a search was then made for secondary brain abscess but none was found. The brain abscess cavity was packed with gauze. She died eight hours later, after a period marked by great restlessness and stupor from which she could be aroused; she recognized and conversed with the physicians two hours before she died.

Anatomic diagnosis (autopsy limited to examination of the head): Acute diffuse suppurative leptomeningitis, abscess of the left temporal lobe, localized left temporal leptomeningitis interna and externa, left mastoid operation with operative opening into the left middle fossa with drainage of the temporal abscess.

#### BIBLIOGRAPHY.

1. Urbantschitsch: *Lehrbuch der Ohrenheilkunde*, 1910.
2. Dickie: *Annals of Otology, Rhinology and Laryngology*, Sept., 1922.
3. Henius: *Acta Oto-Laryng.*, Vol. 5, Fas. 3, 1923.
4. Koerner: *Die otitischer Erkrankungen des Hirns*, 1908.
5. Macewen: *Pyogenic Diseases of the Brain*, 1893.

6. Freund: Arch. f. Psych., 20, 1889.
  7. Oppenheim: Nervenkrank., 1908.
  8. Ballance: Surgery of the Brain, 1908.
  9. Blau: Encyk. d. Ohrenk., 1900.
  10. Mygind: Ugeskra f. Laeger, Band. 80, 1920.
  11. Sowers: Temporosphenoidal Abscess. E., E., N. and T. Monthly, Dec., 1922.
  12. Eagleton: Meningitis. Laryngoscope, 32 (Jan.), 1922.
  13. Plaut and Schottmuler, quoted by Strauss: Am. J. M. Sc., 154 (Nov.), 1917.
  14. Boot: Brain Abscess of Otitic Origin. Trans. Am. Ac. Oph. and Oto-Laryn., 1920.
  15. Lemaltre: Annals of Otology, Rhinology and Laryngology, March, 1920.
  16. Boenninghaus: München. med. W., 48, Mar. 24, 1922.
  17. Starr: Medical Record, Mar. 10, 1906.
- 4100 WEST MADISON STREET.



LXXXVI.

INTRANASAL SYPHILIS \*

By CARL H. CHRISTOPH, M. D.

CHICAGO.

A recent discovery of several unusual cases of tertiary nasal lues prompted a review of the cases reported in the last five years. There is an increasing recognition, in the literature, of the role of syphilis in obscure nasal conditions. While the importance of syphilitic reactions in the upper air passages has long been admitted by all clinicians, each report of such finding is usually expressed as a discovery and a surprising one. This, despite the fact that Fichenbeck,<sup>1</sup> in a series of 235 cases, found the nose involved 82 times. In these cases the disease could not be demonstrated in any other organ. Ballenger<sup>2</sup> states that the nose is involved in three per cent of all cases of syphilis, and Guthrie<sup>3</sup> remarks that lues appears almost daily in the otolaryngologist's practice. Others make similar statements as to its relative frequency. Geiger<sup>4</sup> states that syphilitic nose and throat cases appear frequently in clinical and private practice. Roy<sup>5</sup> and Schmidt<sup>6</sup> agree that obscure specific manifestations in the nose and throat are more frequent than was formerly believed. However, it should be borne in mind that the degree of frequency varies according to the stage under consideration. (I have found that syphilis is not as common in private as it is in dispensary practice.)

Reviewing the subject generally before taking up the three stages in detail, it may be said, first: as to the mode of infection. Syphilis is not essentially a venereal disease, at least ten per cent of infections being extragenital.<sup>7</sup> The nasopharynx has been primarily infected by the eustachian catheter,<sup>3</sup> and a number have been reported infected by other improperly sterilized instruments. Other means—toilet waters, snuff boxes, lingual caresses, kissing, contact of genitals with

\*Read before the Chicago Laryngological and Otological Society, April 7, 1924.

face. Physicians are occasionally infected by picking the nose.<sup>1</sup>

Concerning the time of the appearance of nasal manifestations, the following may be said: They may appear anywhere from one to thirty years after the original infection. Of course, in the hereditary form of the disease, the manifestations usually occur shortly after birth or at puberty.

The site of gummatous infiltration is usually the septum or turbinates. This infiltration is rarely observed clinically, because patients do not consult the laryngologist until the appearance of the stage of softening and decay.

In character, the hard chancre of the nose is usually flat and of moderate size. In the interior of the nose, the chancre is apt to be slightly red or greenish in color, covered with pus. Its appearance, according to Sauer, is that of a fungoid mass, but has the consistency of cartilage. It bleeds easily and the surrounding mucous membrane is more or less inflamed and swollen.

Syphilitic lesions of the nose are similar to those in other organs.<sup>8</sup> The nerve lesions found about the nose are those involving the olfactory, with loss of smell, and those of the fifth and vasomotor nerves.<sup>9</sup> If the condition be neglected, the patient may be left with the characteristic deformity known as "lorgnette nose" as a result of cicatricial contraction during healing.

Schmidt, of Copenhagen, after a series of long misinterpreted cases, states that he learned to suspect syphilis in all cases of disease of the accessory nasal sinuses. Of his 81 cases of sinusitis, over 17 per cent were syphilitic, and of these 9.9 per cent had syphilis of the sinus alone. Graham reported a case wherein, after opening all the sinuses, he later found a positive Wassermann, and mercury rubs gave relief.

In general, the symptoms are first and foremost a nasal obstruction, resulting in a sensation of "stiffness," which later is the symptom that, according to Graham, sends the patient to the doctor. This is not the stiffness arising from the vasomotor disturbances of asthma and hay fever. The appearance here is distinctly different and easily recognized at a glance, with its ashy color. In syphilitics there is a bluish cast to the mucous membrane, and this bluish, ashy tint and

the swelling may extend to the mouth, throat, larynx and bronchi. While I have found that the hypertrophic rhinitis of hay fever and asthma is recurrent, soft and spongy and easily reduced by cocain or adrenalin, the syphilitic type is constant, entirely filling one or both nasal cavities and not influenced by ordinary methods of treatment.

Practically all reports of cases mention this nasal obstruction. The turbinates are large. As Schwartz<sup>8</sup> puts it, patients complaining of chronic obstruction in breathing, in whom there is found a hypertrophied turbinate, polypi, deflected septum with thickening, or a combination of these conditions, should be carefully examined and those with a thickened septum should be suspected of syphilis.

Accompanying this obstruction there is usually a bloody, foul smelling discharge. Some cases have an enormous quantity of ropy secretion discharge which may actually be pulled from the antrum.<sup>9</sup>

When the lesion involves the skin of the nasal vestibule there may be more or less pain in the intraorbital region,<sup>1</sup> for pain in lues is, as a rule, referred to other parts of the head than the nose. Pain due to enlarged turbinates is a heavy pain over the eyes, being seldom felt on the inside of the nose itself.<sup>9</sup> In some cases there may be considerable headache, especially nocturnal headache.<sup>4</sup>

These patients "catch cold" easily, hence obscure rhinologic conditions which resist ordinary treatment are likely to have a syphilitic foundation.<sup>5</sup> Graham gives the case of an eighteen year old girl who had lost much of her schooling because of her almost continuous "colds." Her Wassermann was 3 plus. Antisyphilitic treatment cured her "colds."

Other reported symptoms are: Enlargement of the submaxillary and sublingual lymphatic glands<sup>1</sup> and a constant burning sensation in the nose.<sup>4</sup> Most of Geiger's patients had patches, ulcers or gummas. A few had moderate destruction of bone. He cautions against disregarding sores and patches around the mouth and throat, as spirochetæ can be demonstrated from smears from them. The development of gummata, according to Guthrie, is characterized by nasal obstruction and pain—sometimes severe. As Salinger sums it up, gummata of the septum usually produce marked nasal ob-

struction, profuse mucous discharge and frequent headache.

When discovered early the condition is amenable to treatment and results in most cases in restored structure and function.<sup>10</sup> As Schwartz remarks, prognosis under antisyphilitic treatment is good, as regards gummata, fatty degeneration and absorption taking place. Without treatment the amount of infiltration of the perichondrium and periosteum may become extensive and marked necrosis may follow—with loss of the bony and cartilaginous portions of the septum and in some cases suppuration of the accessory sinuses.

The diagnosis in such cases is vital, as an aid in interpreting other symptoms and because here the disease may endanger life or may result in permanent disfigurement, resulting in loss of voice and regurgitation of food through the nose, etc.

As Sauer points out, although laboratory findings are a great aid, they should not be relied on entirely. Clinical evidence should outweigh even a negative Wassermann.<sup>5</sup> Sauer states that ordinarily no reliance can be placed on examinations of the nasal secretions, because so many spirilla exist in the upper air passages that it is impossible for the laboratory specialist to differentiate between them and that these cases must be studied from other angles. I cannot entirely agree with this statement. While it may be difficult to find the spirocheta pallida in the nasal secretion, it is always possible to differentiate it from other spirilla by means of the dark field. An expert laboratory technician should be able to find the spirochete in a syphilitic ulcer or broken down gumma in at least 75 per cent of cases.

As repeatedly stated in various reports the diagnosis may be difficult—particularly in differentiating a primary lesion from a malignant tumor. Indeed, as Sauer says, diagnosis cannot often be made until the secondary appears. Geiger found that most cases of this type coming under his observation had not been thought luetic—at least the nose and throat condition had not been suspected. In fact, in nearly all, no inquiry had been made into that phase.

Good light is essential to careful examination, because of the peculiar and often obscure places the disease selects in the nose and throat. The characteristic color of the mucous membrane, already mentioned, should arrest attention. In

many luetic persons this membrane has a bluish tinge, a thick, dusky, bluish red appearance, and the application of adrenalin and cocain does not cause this tint to disappear.<sup>9</sup>

If the sore has continued several weeks, syphilis should be suspected. If rhinitis is persistent, after polypi and septal and sinus deformities are ruled out, think of syphilis.<sup>4</sup> All cases of ozena and like nasal conditions or dystrophies should receive most critical examination. A thickened septum with a convexity on both sides should not be operated until gumma is ruled out. This may be syphilitic. If operation be performed it is often followed by a breaking down of the entire septum.<sup>8</sup>

A perforation through the septum, with no history of operation most often speaks for syphilis, especially posterior, although it may be due to constantly picking the nose, to an atrophic condition, to diphtheritic or tubercular infection. Gumma of the septum should be differentiated before perforation, with its attending symptoms.

The gumma itself is frequently overlooked. In the nose, it is found in the septal tissues on the bony framework or on the alae.<sup>4</sup> In some cases the gumma breaks down rapidly; in others, it remains about the same for some time.

In making a differential diagnosis it should be remembered that syphilitic perforations of the nasal septum affect the bony tissue, in contrast to perforation from other causes.<sup>3</sup> Malignant disease is usually unilateral and begins after middle age. Nasal syphilis is differentiated from lupus by its more rapid destruction and by the absence of the "apple jelly" lupus nodules on the skin.

So much for the general considerations of intranasal syphilis. Primary lesions of the nose are comparatively rare. Sauer recalls only three cases. Buckley reported 95 in 9058 cases and four French observers reported only 2 cases in 2244. In 10,000 cases of primary syphilis, the chancre was extragenital in 850 and of these 75 per cent were in the region of the head—mostly in or about the throat and mouth.<sup>11</sup>

Basile<sup>12</sup> reported three cases of primary lesion in the nose. In the first case, a man of thirty, it assumed a diphtheroid erosive form, with intense headache and profuse nasal secretion, all on the left side. He derived no benefit from two

injections of diphtheria antitoxin. The appearance of skin lesions elsewhere three weeks later made a diagnosis of syphilis possible.

His second case was that of a girl of twelve. She appeared to have a neoplasm in her nose. On excising and examining it, its syphilitic nature was discovered. She had charge of an infant which was found to have inherited syphilis.

As to the site of primary nasal lesions: In Geiger's case the primary sore was on the septum, nearly at the perpendicular edge, therefore high up and back. Jaenicke<sup>13</sup> reported one case with the primary sore localized at the entrance of the nares—an undoubtedly rare situation. The man had suffered a slight trauma to the nose. Slight tumefaction followed. One month later a fissure developed at the entrance of the left nostril. Despite cauterization an ulcer finally developed. The Wassermann was 4 plus. The infection was probably from an intermediate source and found a fertile field. Thompson<sup>14</sup> reported a primary lesion in the nares, characterizing chancres in this locality as "medical curiosities." According to Basile, the syphiloma is generally found on the outer wall of the septum. The syphiloma subsides as the disease approaches the secondary stage, and there is none of the destructive action of a tertiary nasal lesion.

As a rule, primaries of the nose are caused by picking the nose with an infected finger.<sup>4</sup> The Wassermann reaction is seldom positive with these initial lesions,<sup>12</sup> thus special attention must be given to the clinical symptoms.

An initial syphilitic lesion may take a form suggesting impetigo, and, while extremely rare, it is difficult to recognize. The rapid development precludes cancer. There is usually a tendency to anemia, and headache on the side affected. The presence of a few enlarged glands, hard, elastic and indolent, in the submaxillary region is suspicious.

Secondary lesions occur more frequently in the nose than is ordinarily admitted, as they are often overlooked by patient and doctor.<sup>15</sup> Sauer does not recall having seen any secondary lesions in the interior of the nose.

The earliest manifestation is a syphilitic "catarrh" or specific coryza. It may occur alone or with a syphilitic pharyngitis or laryngitis. This syphilitic coryza differs very little from

an ordinary cold. It may be limited to one side and the symptoms be not so stormy. In differentiating between it and an ordinary cold, it is noted that instead of a diffused redness there may be only localized areas of erythema. The chief difference is in the duration of the process, the specific coryza lasting much longer. If acute rhinitis does not yield to ordinary treatment, suspect lues and look for other signs.

In children secondary syphilis is a disease of infancy. It takes the form of a persistent nasal catarrh—"snuffles," coryza syphilitica neonatorum—and is easily recognized.<sup>3</sup> The child has a disagreeably persistent nasal discharge, snores and is a mouth breather. This interference with nasal breathing, in turn, interferes with the nourishment of the infant.

Many luetic children are operated on for adenoids, and with harmful results, because the underlying disease is not recognized. A child with Hutchinson's triad—keratitis, peg teeth with notches, fissures and stellate scars about the mouth—should not be operated upon, but treated systemically. Before operating on a child a Wassermann should be done and an examination for bone lesions made.

Secondaries appear in the nose and throat in the form of erythema or mucous patches. These are rather common, but sometimes hard to see. Being superficial ulcerations, they are often overlooked. The area of hyperemia surrounding them should betray their character. The occurrence of papules or broad condylomata in the interior of the nose is a disputed question. According to Schleich,<sup>10</sup> they are most often found on the septum and on the floor of the nose. They are frequently milky white or grayish white epithelial thickenings with an area of inflammation about them. However, it is conceded that mucous patches alone or with patches in the mouth and on the fauces are comparatively rare. I have seen no cases that could be classed as primary or secondary or that were diagnosed as such, my cases all being confined to the tertiary type.

Tertiary lesions of the upper air passages are not so infrequent that they should fail to be recognized by the general practitioner, who is usually the first to see them.<sup>10</sup> Indeed, tertiary lesions of the nose are common occurrences, according to Sauer, comprising about 4 per cent of the cases. Mauriac<sup>17</sup>

found the nose and pharynx involved 54 times in 237 cases of tertiary lues.

These lesions are not detected, because the primary lesion so far antedates the present complaint that both patient and physician do not see the connection. The period between the initial lesion and the tertiary varies a great deal. Wile found it to be ten years, but Geiger found it to be seven. I found it to be eight to ten years. Often the patient has been told by some doctor that he has been cured. Or the physician neglects to make a thorough examination, or, in making one, fails to see the lesion. In all cases with head symptoms, such examination should be made as a routine measure. General physicians should be more proficient in the use of the head mirror. Thorough routine examination of the nose and throat in all new cases will give any physician sufficient skill to detect pathologic changes in these narrow passages. Local examinations are of vast importance, because it is only by inspecting the lesion that one suspects syphilis. Once seen, the lesion—whether its true nature is recognized or not—will lead to taking a Wassermann test, and thus both physician and patient will be saved from an error in diagnosis.

The common form of nasal syphilis is the tertiary gumma.<sup>3</sup> Gummata are seen more frequently in the nose or hard palate than in the pharynx. In my cases the lesions were in the septum and inferior turbinates.

These lesions were seen in different stages. The gumma involving the inferior turbinate, completely blocking the nose, was usually unilateral—a smooth hard inflamed mass, which nothing would reduce. Others showed a superficial ulceration in which spirochetæ were found. The gumma in the septum showed a bilateral thickening, usually varying in size from a pea to a hazelnut. Some cases were seen later after perforation had taken place.

Seen early, the gumma of the septum appears usually over the vomer as a smooth circumscribed swelling, covered by a generally inflamed mucosa. It may appear anywhere along the upper respiratory tract, from the vestibule of the nose down to the subglottic space, in the early form as a gumma or a chondritis, and later as an ulcer, circumscribed, deep and destructive, or superficial and serpiginous. The favorite seat



of gumma is the bony septum, although Salinger remarks that they are frequently found also on the hard and soft palate, the posterior wall of the pharynx, the epiglottis and its folds. Usually there is no sharp outline to the infiltration, but a gradual fading into normal tissue. Gummatous infiltrations involve the mucous membrane, cartilage and bone.

Diagnosis is usually easy, but at times may be difficult. Any ulcer involving the bone and any sequestra point to syphilis. Bone necrosis is an important diagnostic feature: black sequestra are often exfoliated. Perforations of the bone of the septum are usually syphilitic. Yet many tertiary stages are frequently undetected and hence improperly treated and the patient is thus exposed to irreparable harm.

Differentiation in perforation: Lupus may destroy the cartilaginous portion but not the bone. Perforation of the septal cartilage may be luetic but is usually due to a simple perforating ulcer.<sup>1</sup> In tuberculosis the bone is not involved. Bone necrosis at once differentiates the disease from ozena.<sup>3</sup>

Often the swelling is bilateral and may resemble a hematoma, from which it is differentiated by the history of an indefinite onset, absence of trauma and the localization of the swelling. A hematoma of the septum produces a more diffuse bilateral swelling, taking in the cartilaginous as well as the bony portions.<sup>10</sup>

The swelling may be mistaken for a septal deflection, but should be distinguished from that by the absence of the angularity which characterizes most deflections, as well as the lack of the usual concavity of the opposite side of the septum.

At the beginning the subjective symptoms may be quite indifferent and are often mistaken for chronic coryza or hay fever. After a time the discharge from the nose becomes more purulent, foul smelling and bloody, with loss of the sense of smell. Small crusts form in the nose and are difficult to expel. Later the foul odor becomes more apparent, and this odor is considered by many as characteristic of syphilis. Pain in the nose and over the bridge may be quite severe at times. It may radiate to the ears and to the teeth. Frequently bony sequestra are blown from the nose or may pass out through the nasopharynx. In neglected cases extensive destruction may involve the bone of the entire septum, turbinates and hard pal-

ate. But considerable time may elapse before there is a breaking down. This last usually occurs in the center of the infiltration, leaving an ulcer with a depth greater than its diameter. This ulcer has well defined margins and is covered by a sloughing membrane. The headache is more or less constant and there is a foul, profuse, mucopurulent discharge.

When undiscovered and neglected the results are dire. A perforation of the septum results, leading in its last stages to external deformity—a sinking in, the well known saddle nose, although it should be remembered all saddle noses are not syphilitic. Abscess following injury may cause it, too. It is especially in hereditary syphilis that this great disfigurement so often occurs.<sup>3</sup>

These deformities resulting from tertiaries about the nose and throat are very apparent and easily recognized. Not so much loss of tissue is seen now as formerly, but Salinger feels this is due not so much to early diagnosis as to intensive treatment.

From all these signs, symptoms and hints as to differential diagnosis, it is evident that there is little excuse for misinterpreting or overlooking the basic element in these nasal conditions. The very obscurity of the trouble, the very difficulty of diagnosis, should prompt one to more careful local examinations, to the taking of a Wassermann test in all suspicious cases.

#### REPORT OF CASES.

Case 1.—M. C., female, aged 31, came into the office complaining of difficulty in breathing through the right side of the nose and pain over the right eye and right frontal region.

Examination showed an enlargement of the right inferior turbinate, entirely filling the nasal cavity. The hypertrophy was of a bony consistency and could not be reduced by cocain or adrenalin. There was a slight nonfetid serous discharge. Transillumination and X-ray showed a marked cloudiness of the right frontal and maxillary sinuses. The temperature was 99 and the patient had a general malaise. The differential diagnosis lay between lues and a neoplasm. The Wassermann was 4 plus and the patient immediately was put on antispecific treatment. At the present time the tumefaction

is already one-third reduced, and the patient is able to breathe without difficulty. The antrum also is rapidly improving.

Case 2.—R. B., age 22, female, came into the office complaining of bilateral nasal obstruction of an alternating character. The septum showed a bilateral bulging posteriorly and superiorly, but the enlargement seemed to be bony rather than due to a soft tissue hypertrophy.

X-ray pictures of the sinuses were negative. A submucous resection was performed. Following the operation there appeared a sloughing in the upper posterior portion of the septum with a foul discharge. At the same time there appeared a falling of the nose similar to a saddle nose.

The Wassermann was 3 plus, and on specific treatment the patient improved rapidly, although the deformity remained.

#### DIFFERENTIAL DIAGNOSIS.

Ernst Wodak, *Arch. f. Laryngol. u. Rhinol.*, 34:194, No. 2-3 Berlin, 1921.

Clinical Picture. Headache, fetor, neuralgia, temperature, etc., present in both tuberculosis and lues. Pirquet of no value. Wassermann, guinea pig injections of value, considering above—may be very difficult to differentiate.

Primary Syphilitic Lesion in Nose.—Pietro Ramogini. *Gior. ital. d'mal. nose.* Milan, 63:789, No. 3, 1922. Small swelling, ulcerated at summit, covered with adherent, lardaceous or pseudodiphtheritic exudate, pains radiating to head or orbit, complete or partial occlusion of nasal cavity, abundant flow of mucous, sometimes blood tinged, swelling cheek, ala, submaxillary glands near greater cornu of hyoid bone, preauricular or parotid glands swollen. Chancre prolonged course in nose, may last into secondaries. Due to location, blowing, picking, mucus, dust and crusts, cicatrize 40 to 50 days after first appearance. Sequel—synechia.

Unusual case of syphilis in nose.—P. Copacearm, *Spitalul.* Bucharest, 42:209, July-August, 1922. Woman, 68; ulcer of nose 1½ month duration. Diffuse hard infiltration of ala nasi on inner side of which erosion covered with gray yellow crust extending to nasolabial fold. Eroded angle of mouth, 4 papules, upper one eroded. Later roseola developed. Wassermann positive. Primary lesion mode of infection not known.

Syphilitic Polypi of Nose.—Jacques, *Ann. d. mal. de l'oreille, du larynx, etc.* Paris, 41, 1114, Nov. 1922. Slow hyperplasia, resembling polypi; diffuse, grayish yellow vegetation, slight mobility and granular or uniform surface. Combined with accessory sinus suppuration not yielding to surgical treatment.

## REFERENCES.

1. Sauer, W. E.: Syphilitic Reactions of the Ear, Nose and Throat; *Am. J. Syphilis*, 1920, 4, 430.
2. Ballenger: Cited by Sauer.
3. Guthrie, D.: Syphilis of the Throat, Nose and Ear: Its Diagnosis and Treatment; *Practitioner*, 1920, 104, 131.
4. Geiger, A. H.: Manifestations of Syphilis in the Nose and Throat; *Ill. Med. J.*, 1919, 36, 233.
5. Roy, D.: Report of Interesting Nasal Cases Due to Syphilis. Remarks on Obscure Syphilitic Nasal Symptoms; *Ann. Otol., Rhin. and Laryn.*, 1917, 26, 967.
6. Schmidt, V.: Syphilitic Disease of Nasal Accessory Cavities; *Hospitlaestid*, 1920, 63, 544, 553.
7. Buckley: Cited by Guthrie.
8. Schwartz, E. M.: Gumma of Nasal Septum; *N. Y. M. J.*, 1923, 118, 289-290.
9. Graham, H. B.: Frequent but Neglected Evidences of Syphilis from the Side of the Nose, Accessory Sinuses and Ear; *Am. J. Syphilis*, 1919, 3, 26.
10. Salinger, S.: Tertiary Syphilis of Nose and Throat; *Ill. Med. J.*, 1919, 351, 126.
11. Fournier: Cited by Guthrie.
12. Basile, G.: Initial Syphilitic Lesions in Nasal Fossas; *Poli-clinica (sez. Prat.)*, 1918, 25, 510.
13. Jaenicke, K.: Primary Syphilitic Nose Infection; *J. A. M. A.*, 1921, 77, 1889.
14. Thompson, L.: Syphilis, 2nd Edition, p. 72-73.
15. Tissier: Cited by Sauer.
16. Schleich: Cited by Sauer.
17. Mauriac: Cited by Sauer.

## ADDITIONAL REFERENCES.

- Hays, H. M.: Syphilitic Manifestations in Nose: Report of Two Cases; *J. A. M. A.*, 1921, 76, 1575.
- Ravogli, A.: Syphilis of the Face; *Lancet-Clinic*, 1909.
- Fanoni, A.: Observations on Nasal and Intranasal Syphilis; *Post-Graduate*, 1907.
- Beck, J.: The Wassermann Reaction and Salvarsan in the Diagnosis and Treatment of Syphilis in Ear, Nose and Throat Affections With Special Reference to the Auditory Nerve; *Ann. Otol., Rhin. and Laryn.*, 1912.

LXXXVII.

CELLULITIS OF THE ORBIT IN INFANTS AND  
CHILDREN WITH A REPORT OF TEN CASES.\*

BY KENNETH A. PHELPS, M. D., F. A. C. S.,

MINNEAPOLIS.

For the reason that I disagree with the predominant belief that orbital cellulitis is more prevalent in adults than it is in infants and children, I am limiting my observations in this paper to the latter. This is exemplified in my own experience in that, during a comparatively short time, I have seen ten of these cases. I am firmly of the opinion that children are very much more subject to orbital cellulitis than has been recognized heretofore. A recent encyclopedia of ophthalmology,<sup>1</sup> competently edited, contains the following assertion: "Due to the undeveloped condition of the sinuses in children, few cases of orbital cellulitis are recorded under nine years of age." With all due respect for this opinion, I believe it should be modified; as nine of the cases I have just referred to were children who had not yet attained nine years of age.

The most frequent and usual cause of orbital cellulitis—I am limiting my remarks to children—is often overlooked, and this by experienced physicians. It is my belief that practically all cases of orbital cellulitis have their origin in disease of the nasal accessory sinuses. Nine of the cases that I am about to detail to you fall within this category. There are several important anatomic conditions of the sinuses and the orbit in children which favor and contribute to the development of orbital complications of sinusitis.

(a) First, regarding the bones: there may be congenital dehiscences along the ethmomaxillary suture, in the lamina papyracea, or in the orbital wall of the maxilla.<sup>14</sup> The orbital bones are softer in children, ossification not being complete

\*Read: American Academy of Ophthalmology and Oto-Laryngology, Washington, D. C., October 17, 1923.

until the sixteenth year. The sutures are ununited until the sixth year.

(b) The orbital periosteum is thin and delicate; it not only lines the openings into the orbit, but forms one of the coverings of the nerves and blood vessels passing through them, thus becoming continuous with the dura mater at the superior orbital fissure.<sup>1</sup> This periorbita also extends into the nose through the lacrimal duct. Schaeffer<sup>2</sup> is authority for the statement that at times the lacrimal sac and the mucous membrane of the ethmoid cells are in contact, thus making ethmoid infection and lacrimal sac infection hard to differentiate. In fact, he says there are cases recorded of orbital cellulitis originating in dacryocystitis. The periorbita is easily detachable from the surface of the bone, but it is adherent at the sutures where in early life, it is continuous with the periosteum covering the opposite sides of the bone.

(c) The sinus mucosa is in most intimate relation with the osseous tissue so a slight inflammation of one produces a change in the other. Armstrong and Dean<sup>3</sup> believe that in cases of equal clinical severity with equally marked changes in the mucous membrane, there is much more apt to be involvement of the underlying bony wall in the child than in the adult.

(d) There is more profuse development of the lymphatic and vascular system than in the adult. Infection from the sinuses may enter the orbit along the veins without any bony perforation. Thus the infection in a child's nasal sinus has a much easier path to the orbit than it has in the adult.

The pus does not in all cases perforate into the orbit: it may be guided anteriorly beneath the periorbita until it points through the eyelid where it sometimes ruptures spontaneously. We are all aware of the frequency of subperiosteal abscesses of the mastoid in children and should realize that the same condition could occur in the orbit as a result of infection in the nasal sinuses. Valero<sup>2</sup>, of Spain, reports a series of such cases this year.

I do not wish to be understood as stating that an infected nasal sinus is the only cause of orbital cellulitis. There are several other contributing factors among which may be numbered, trauma, carious teeth, osteomyelitis, erysipelas, menin-

gitis, brain abscess, the exanthemata, metastasis or actinomycosis, but these causes are infrequent when compared with nasal sinusitis.

The first symptom to be noted of orbital cellulitis is sudden edema of the eyelids, which in all probability, closely follows the rupture through the periorbital. The edema may involve the lids of one eye or of both. It usually increases so rapidly that within twenty-four hours the eyelids are so edematous that they can with difficulty be opened. The edema may be accompanied by pain in the eyes, radiating to the forehead.

Chemosis is usually present and it may be pronounced enough to cover the entire cornea. Frequently chemosis may give a valuable clue as to the exact location of the pus in the orbit: if the chemosis is nasal, then the pus will be found in the nasal portion of the orbit; if it is temporal, then look for the pus in the temporal section of the orbit. Following chemosis, proptosis manifests itself, but if the case is seen early enough it is possible to forestall it by proper treatment. But when the proptosis is evident, the position of the eye may be a valuable aid in locating the pus. Limitation of extraocular movement is another and frequent symptom, but a history of diplopia, associated with it, is very difficult to obtain in children. Examination of the fundus usually discloses dilated retinal veins. There may be cases of indentation of the globe simulating detached retina, choked disc, optic atrophy, optic neuritis, retinal hemorrhages, or retrobulbar neuritis,<sup>6 7</sup> but I have never happened to observe any of them.

The general condition of the child appears serious; the temperature high, there is a marked prostration and signs of sepsis are frequently present.

At times the physician can secure a history of colds or of infectious disease, but one of sinusitis is very difficult to obtain from small children.

Children seldom complain of nasal discharge and the diagnosis depends almost entirely upon the examination. As I have pointed out elsewhere,<sup>8 9</sup> the most valuable single diagnostic aid is the X-ray; but the value of the X-ray in the diagnosis of the orbital condition is limited. Most of my cases disclosed under the X-ray pansinusitis of one side, and in each case this was confirmed and verified at the time of

operation. *Streptococcus hemolyticus* was found in every case in which a culture was made. The importance of an early diagnosis of sinusitis is shown by the fact that, in that stage prevention of orbital complications can often be accomplished and frequently by nonoperative measures; but once the orbital contents are involved, I believe that the condition should be treated as an emergency and an immediate operation performed. In the case of very small children, intranasal drainage of the orbit is not satisfactory. If free pus has been formed in the orbit it should be drained externally at once, as the orbital tissues disintegrate very rapidly, forming one purulent mass which may lead to total loss of the eye.<sup>10 11</sup> An external operation should be performed with the following technic:

With the patient anesthetized, the operator sits at the head of the table, which has been lowered to such a degree that the patient's head rests almost in the physician's lap. Some form of a good headlight should be used and I favor the head-mirror. The nasopharynx is packed off. I have found it advisable to place a piece of adrenalin soaked gauze with a thread attached thereto in the middle meatus. This serves as a landmark and it cannot be lost or forgotten if the string is left hanging out of the nose. A curved incision is made almost identical to that used in the extirpation of the lacrimal sac. Should the sac be encountered it is lifted from its groove. The periosteum is elevated from the lacrimal bone and the os planum. A perforation is usually discovered through the lacrimal bone or the ethmoid, though the antrum may be the sinus to blame.<sup>12</sup>

A nasal septum retractor is very useful in holding back the eyeball and Tenon's capsule. Pratt's curette is the best instrument I have found to break down the tiny ethmoid cells and to establish a free drainage into the nose. The antrum should be opened from above in very young children, but through the inferior meatus when possible and the frontal or sphenoid explored when indicated. Drains are left in the nose from the sinuses and the incision should be left almost entirely closed. Adenoids should be removed. In twenty-four hours the drains are removed. In case exophthalmos is marked, considerable care should be taken of the cornea, as repeated



dressings of the wound will expose it to possible trauma, which might eventuate in ulcer, with possible loss of the eye.

I have a number of illustrative cases, some of which I will report in detail, whereas others will be given with less particularity. The first is that of a boy three years old referred to me by Dr. Max Seham. His lower eyelids were red and swollen following a cold. There was no chemosis or exophthalmos present, but there was considerable nasal obstruction, associated with purulent discharge; the temperature was 100 and the general condition of the child seemed good. Dr. Seham prescribed the use of a nasal suction pump, which gave relief and he seemed to be doing well. After about ten days the right upper eyelid suddenly became edematous. The edema rapidly increased to such an extent that it was absolutely impossible for him to open his eyes. There was a copious discharge from the right nostril. Temperature was 102½. I was called in and found that his middle meatus contained pus. Transillumination disclosed that the right antrum was dark. An X-ray examination of the sinuses made by Dr. Allison revealed a clouding of the right ethmoids and antrum, with absent frontal sinuses. The eye was quiet, no chemosis, proptosis or limitation of movement; but the patient complained of pain when he rotated his eye. The fundus was negative. A diagnosis of acute sinusitis, with early cellulitis of the orbit, was made and an immediate operation was performed following the technic suggested above.

A perforation was found through the ethmoid bone, but no free pus was found in the orbit. The ethmoid cells and antrum were filled with thick pus. These cavities were curetted and drained through the nose, the wound being completely closed. In twenty-four hours the temperature was normal and in two weeks a complete recovery resulted.

My second case is that of a ten year old girl, who complained of pain in the right eye, accompanied by swelling of the right upper eyelid. The pain started about twenty-four hours before her admission to the hospital, followed by swelling twelve hours later. No history could be secured of any contagious disease or of any other illness preceding the present trouble. There was no history of nasal discharge and the child did not experience headaches. At the time of admission

the temperature was 103, the pulse was 116, and respiration was 16. The child had a toxic appearance and both right eyelids were very edematous and red, although the left lids were but slightly puffy. Chemosis was very marked in the right eye and proptosis was so advanced that there was very little power of rotation in any direction; but no diplopia was complained of. Vision was 20/30 in the right eye and 20/20 in the left. The right cornea was clear and the pupil reacted readily. The fundus revealed dilated veins, but no other pathology was in evidence. Examination of the nose revealed no pus, perhaps because it was draining into the orbit,<sup>5</sup> but in an X-ray examination, all of the right sinuses were found to be cloudy. A diagnosis was made of orbital cellulitis, but it was hoped that a stab incision through the upper eyelid would produce sufficient drainage to give relief. **This was** found to be insufficient in that it drained the orbit with no effect upon the sinuses. The next day a radical external operation was deemed necessary, performed, like my first case, after the technic referred to above. Thick pus was found, under pressure, in the frontal, all the ethmoids and the antrum. Drainage of these cells, and of the orbit, also, was brought about through the nose, the external wound being closed. It was necessary to treat the child three months before she recovered, during the course of which a metastatic abscess developed in the right ankle as well as one in the scalp, both of which required incision and drainage. The eye itself was injured by reason of the dressing coming into contact with the cornea and producing an ulcer which, in spite of treatment, progressed to perforation, adherent leucoma resulting. The sinuses cleared up quite completely.

The next case is that of an infant a year and a half old. A week before admission to the hospital, swelling was noticed in the left cheek, which traveled to the outer half of the upper eyelid. The eyelid rapidly increased in size, becoming very red and inflamed. The family physician, upon being consulted, incised the eyelid, but located no pus. The infant was then taken to the hospital. His temperature was 98.6. The left eye was pushed forward, downward, and outward. The upper bulbar conjunctiva was chemotic; the cornea was ulcerated; the white blood count was 42,000. Examination of the

nose disclosed no pus and no external excoriation. Upon the sinuses being X-rayed, a left pansinusitis and a cloudy orbit were found. Diagnosis of orbital abscess and sinusitis was arrived at and a radical external operation was performed which confirmed the diagnosis, as the antrum, ethmoids and sphenoid all contained thick pus. We were unable to check the ulcer of the cornea, which ultimately perforated resulting in loss of the eye. During the course of the thirty day treatment that followed, a metastatic abscess developed in the right ankle, which required incision and drainage.

My next four cases are so similar in all respects that they can properly be grouped together. One patient was five months old, two were six years of age; the other was seven. In each case there was sudden swelling of the lids, chemosis, exophthalmos and temperature. X-ray of the sinuses disclosed that the ethmoids, antrum and orbit of the affected side were all involved; and this was true of each of these four cases. In each case the typical operation was performed and a perforation found into the orbit from the ethmoid cells, with free pus in the orbit.

All recovered completely without visual defects. My next case resembles these four with the exception that during the course of the operation cerebrospinal fluid escaped through the cribriform plate, although no deleterious results eventuated.

Case nine is that of a two year old girl referred by Drs. Huenekens and Moriarity. She was in her second week of scarlet fever when the left eyelids suddenly became red and swollen. She had considerable headache and much purulent nasal discharge from the left nostril. Her temperature was 101. Examination revealed that the tonsils were well removed, much pus was seen running down the postpharyngeal wall. The left preauricular gland was enlarged, also the left cervical glands. Much pus could be obtained by suction of the left nostril. The eye showed no chemosis, proptosis or limitation of motion, vision normal, pupil regular and reacted, fundus showed slight engorgement of the retinal veins. A diagnosis of acute sinusitis was made, and the suction pump prescribed. Great improvement followed use of suction, and the lid edema disappeared in forty-eight hours. Four days later the symptoms returned with chemosis starting at the caruncle and

bulbar conjunctiva slightly edematous. Operation was advised but refused. Hot compresses were used and in the morning the eye was full of pus and the swelling had gone down. Under the upper lid near the inner canthus the point of rupture was found. For a month the child progressed well, when a sudden recurrence of all the symptoms appeared. Chemosis was more marked, temperature 102 and the child seemed much worse generally. Operation was performed as outlined above. A perforation through the ethmoid bone was found but no pus was in the orbital cavity. This was a subperiosteal abscess which had produced orbital symptoms due to serous exudation and vascular changes but no actual infection of the orbital contents. The antrum was opened through the inferior meatus and drained into the nose. A large mass of adenoids was removed. The child had very little reaction to the operation and progressed to an uneventful recovery.

The last case that I will present is that of a male child three years of age referred by Drs. Schlutz and Stewart. His first symptom was severe headache, not preceded by a cold or any other illness. The right temporal region was somewhat swollen, but no earache or discharge were noted. At the end of two weeks, this swelling disappeared followed by exophthalmos of the right eye, accompanied by marked chemosis and edema of both eyelids. The temperature was high, but there was neither convulsions nor paralysis. The exophthalmos increased until, at the end of the fourth week, a fistula appeared at the external canthus, discharging pus, which gave considerable relief. During the next two weeks Jacksonian convulsions took place, beginning in the left arm and becoming generalized. During the next month, but little change was noted, the child still complained of severe headache; exophthalmos persisted and the fistula continued to discharge. The patient was brought to Minneapolis to Drs. Schlutz and Stewart who found a rigidity of the neck, with a positive Kernig sign, a positive Brudzinski sign and sluggish knee kicks on the right and normal on the left. The spinal fluid was found to be under but little pressure, clear, contained 12 cells, positive Nonne and a negative Wassermann. White blood count was 15,000, with 61 per cent P. M. N.'s; the urine was negative; the temperature was 99. At that time

I was called in. I found the right upper eyelid edematous, but not sufficiently so to prevent the eye from being opened. Exophthalmos was present, the eye apparently being pushed directly forward. Upon palpation of the globe, no resistance was encountered in the orbit. The pupil was slightly dilated and reacted readily to light; the cornea was clear. Extraocular movements were limited laterally, but were good in other directions. The fundus disclosed dilated veins, without choked disc. A small fistula at the external canthus was discharging pus, through which opening a probe was readily passed without increasing or decreasing the volume of pus emitted. The nasal sinuses, in the X-ray, were found to be clear, although the orbit was cloudy. The nose, throat and ear examinations were negative. It was deemed advisable to explore the orbit, which was done under a general anesthetic.

Necrotic bone was found at the apex of the orbit, but no abscess cavity was encountered. The orbital roof was intact. The patient reacted very slightly to this exploration and there was no appreciable change in his condition. Early in the morning of the postoperative second day, the child was awakened by a severe headache, which was not susceptible of control by morphin. He was entirely rational, rolled over in bed, and died. A postmortem was performed by Dr. Charnley McKinley, of the University of Minnesota. A large temporal lobe abscess was found which had perforated the orbit at the superior orbital fissure, remaining external to the periorbita, extending the entire length of the orbit and breaking through at the external canthus. A localized meningitis surrounded the abscess; the middle ear and mastoid contained seropurulent material and the tympanic membrane was thickened but intact.

It is known that a brain abscess can break through the orbit and produce an orbital abscess, but I have been unable to find a report of any such case in the literature. Dandy, in a personal communication, writes that he has never seen such a condition. As the sixth nerve passes through the superior orbital fissure, one can readily see how it could become involved in this case; and the continuity of the periorbita with

the meninges also explains how such an accident could occur without the orbital contents being involved.

In conclusion, orbital cellulitis is more frequent in children than has been recognized heretofore, and this is accounted for by reason of the anatomy and development of the nasal sinuses and the orbit. The most frequent cause of orbital cellulitis is infection of the nasal sinuses. If this is recognized early enough the orbital complications can often be prevented. Radical treatment should be resorted to as early as possible after the diagnosis of orbital involvement is made, thus curing the sinuses, conserving the vision and, in many cases, saving the life of the patient.

#### BIBLIOGRAPHY.

1. Am. Encyclopedia of Ophthalmology.
2. Valero: Arch. de Oftal. Hispano-Am. 23, 261; May, 1923.
3. Schaffer: The Nose and Olfactory Organ, Blackiston, 1920.
4. Oppenheimer: Jour. A. M. A., Vol. 73, No. 9.
5. Dean, University of Iowa: Studies in Medicine.
6. McCullough: Trans. N. Y. Acad. of Med., Oct. 26, 1921.
7. White: Quoted by Dean.
8. Phelps, K. A.: Sinusitis in children; N. Y. Med. Jour., September 22, 1917.
9. Phelps, K. A.: Sinusitis in children; Jour. Lancet, Nov. 1, 1920.
10. Skillern: Accessory Sinuses of the Nose.
11. Hajek: Ztsch. für Hals, Nase and Orheilck., Berlin, 4, 511; April 25, 1923.
12. Scammon, Prof. of Anatomy, U. of Minnesota. (Personal communication).
13. Dean: Ann. of O. R. & L, 1923, p. 285.
14. Whitnall: Anatomy of the Orbit.
15. Vail: Trans. Am. L., R. and O. Sec., 1919.
16. Scammon, Prof. of Anatomy, U of Minnesota. (Unpublished research).

LXXXVIII.

SUSPENDED RESPIRATION IN A CASE OF BRAIN  
ABSCESS.

FREDERICK N. SPERRY, M. D.,

NEW HAVEN, CONN.

This case of brain abscess is related as an introduction to the paper and demonstration of Dr. Henderson. The specimen shown is part of the temporal lobe of the left cerebral hemisphere.

A complete postmortem examination was made showing among other things the following: Chronic osteomyelitis of temporal and parietal bones; sinus of mastoid process, extending from skin to dura; greenish serous exudate in middle ear and mastoid showing Gram positive and negative organisms; chronic leptomeningitis extending to occipital bone; organized thrombi obliterating sigmoid, transverse and superior petrosal sinuses; numerous abscesses in posterior pole of temporal lobe. The largest, 1.5 cm. in diameter, contained about 2 cc. of green purulent exudate showing pus cells, but no bacteria.

The examination also showed chronic mitral endocarditis, fibrous pleurisy and bronchiopneumonia.

With this knowledge of the case I should like to relate a short clinical history. Mr. Clarence Haney was brought to New Haven Hospital May 14, 1923. Family history negative, and personal history, up to seven years ago, is irrelevant.

Seven years ago had purulent otitis media, left side. After a discharge lasting one year a swelling appeared back of the ear, which was incised. Incised wound and left ear discharged pus to the time of admission to the hospital, a period of six years.

One month before admission began to ail. Felt tired, appetite was poor, vomited, projectile in character after meals, and for three weeks had had diplopia. Until a week before this time had no pain. During this week headache, mostly frontal, had been severe. On the day of admission the pain

changed to the back of his head. The pain was constant but worse at night. Vomiting was increasing in severity and patient felt drowsy but could not sleep on account of pain. There had been dizziness.

Entered the hospital at 11 A. M., and had a convulsion shortly after. Both hands were involved, the left being more affected. A chill followed the convulsion but temperature remained subnormal. Temperature 97, pulse 50, respiration 20. blood pressure 112/40. Pupils equal, no nystagmus. Normal reaction to light and accommodation. Ophthalmic examination showed considerable choking of both discs. The margins were indefinite, the veins considerably dilated and tortuous.

No rigidity or pain on flexion of neck. Knee jerks lively and at times are held spastic. Great toes were drawn up in spastic extension. Some ankle clonus on right side.

Lumbar puncture at 5 p. m. Fluid was under pressure and ground glass in appearance. About 15 cc. were withdrawn. Cell count 200, 80 per cent polys. Blood, red count 5,390,000. white count 19,700,91 per cent polys.

In the afternoon respiration assumed Cheyne-Stokes character on several occasions. At 8:15 p. m., gas oxygen, followed by ether, was given and during induction respiration became Cheyne-Stokes in character. Twenty minutes later the ear canal had been cleaned of foul cerumen, and the field of operation prepared, when the patient stopped breathing. The pulse, which had been 52, became 160 and artificial respiration, Sylvester method, was begun together with oxygen inhalation. This was continued for six hours and forty-eight minutes when the heart failed.

After four hours of artificial respiration, decompression operation was done. The dura bulged and when incised the brain also bulged. A canula disclosed pus at a depth of 3 cm. Only about 1 cc. was evacuated. Shortly before death carbon dioxide inhalation was used with no noticeable improvement.

The possibility of using carbon dioxide did not occur to us earlier and I feel that in this case it could not have saved life. We have in it, however, a powerful adjunct. The place it occupies will be shown by Dr. Henderson.



## SOCIETY PROCEEDINGS.

---

### CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

*Meeting of Monday, April 7, 1924.*

THE PRESIDENT, DR. JOHN A. CAVANAUGH, IN THE CHAIR.

DR. CARL H. CHRISTOPH presented a case of

#### **Black Hairy Tongue (*Lingua nigra pilosa*).**

The patient was a man, aged 38 years, who gave a history of two attacks of influenza, frequent attacks of tonsillitis, two frontal sinus infections, bronchopneumonia, and a "nervous breakdown," but who had no gastrointestinal disease and no gingivitis. In January, 1923, following an attack of influenza, he took a few doses of cough mixture which contained codein. Three or four days later he noticed that the tongue was becoming discolored and developing long, hair-like structures. The only subjective symptom was a slight thickness of the tongue.

DR. OTIS H. MACLAY presented a case of

#### **Auditory Aphasia.**

The patient was a boy aged eight years. Birth was normal and at term. He walked and talked at the age of fourteen months. Dentition occurred at the usual time. His parents are living and well.

When two years old he had diphtheria. Antitoxin was injected and recovery followed. Ten days following the acute attack, the boy became suddenly paralyzed so that he was unable to walk. There was paralysis of deglutition, fluids regurgitating through the nose. This latter lasted for about ten days with gradual improvement. While in bed with a paralysis of the lower extremities he cried considerably, especially when he was handled, apparently suffering a great deal of pain. Whenever he was touched or lifted from the bed he shrieked with pain. The motor paralysis improved grad-

ually and after a period of about five weeks the patient was able to walk, although slowly, and his step was neither firm nor certain. He would stumble occasionally. After that time improvement from the paralysis was apparently complete. He was able to walk and run like other children of his age.

Several months after the acute diphtheritic infection with the paralysis that followed, the parents noticed that the boy was inattentive when spoken to. He would look at his mother and watch her speak when she talked to him, and it appeared to the parents that the boy was deaf; for whatever was said to him had to be repeated several times and then he would get an incomplete idea of what was said. As he grew older he was sent to school, and passed in two grades within the past two years with apparent ease. He is able to read, write and do some figuring, but the teacher, like the parents, believed the child to be deaf. Upon examination Dr. Maclay found that the nasopharynx was negative, the ear drums normal, and that the patient can hear a whisper, the high notes of a whistle, and the various forks. Air conduction and bone conduction are normal. He concluded that this was not a case of deafness but that there must be some involvement of the auditory nerve apparatus which is responsible for the condition.

He observed that when speaking to the patient about some subject that is new to him, he did not have to speak in a loud tone but would have to repeat the subject several times in order to make him understand. He would understand at once if gestures were used. It was apparent that the difficulty here lay not in audition but in the comprehension of the meaning of speech. The boy has a good vocabulary for a child of his age. If he became deaf at the age of two years he could not possibly have acquired such a vocabulary, though he occasionally misspeaks words and misnames objects.

#### DISCUSSION.

DR. ALBERT B. YUDELSON said that auditory aphasia, or word deafness, is a condition in which the patient's auditory mechanism is intact, but his auditory word memory is affected as a result of a lesion in the left temporal lobe. Being able to hear but lacking memory for the meaning of words, they

are meaningless to him, and he often uses meaningless words. There is a blocking between the receptors of spoken speech and their central station. In an effort to express himself, the patient forms words of his own. Thus he may misname objects—paraphasia—and utter entirely unintelligible symbols in expressing an idea. Should he attempt to repeat the same thought he may use other symbols equally as meaningless. Nor can these patients repeat words spoken to them or copied to dictation.

There is a variety of degrees in auditory aphasias. Some patients fail to understand anything that is said to them, others understand some things, still others comprehend most words. Likewise, in their effort to express themselves, some patients are not at all aware of the mistakes they make, others are partially aware of them, while still others express themselves quite clearly, and inject their gibberish only occasionally. They cannot explain the meaning of their symbols. The intellect is also affected to some degree in these cases.

The patient presented here had a partial auditory aphasia. Neurologic findings were negative. When spoken to, the boy watches the examiner's lips in an effort to obtain a suggestion of what is said. He listens intently and thinks hard, trying to understand the meaning of the words addressed to him. He appeared to be deaf, but Dr. Maclay said the patient's hearing is good. His hardship in understanding what is said to him is due to defective word memory.

The patient also has paraphasias. He misnames things frequently and coins unintelligible words. He calls a dog's eye "EE-eye" and a thin, long neck of a lean person is "Mars" or "Math" neck. A passenger train made up of many cars is a "Gin-i-gin" and a suburban train is a "Diddle-Diddle." "Araltonose" is the shiny part of a dog's nose and a "E-wheel" is one with a solid tire. "OO-leg" is a table leg with a ridge on the top. It took a long time to find an explanation for these symbols, nor is it at all certain that their interpretation is correct. Were the patient's aphasia of a greater degree his intelligible vocabulary would have been less, and his jargon even more extensive. There did not appear to be any pronounced mental defect in this case.

**\*Paper: "An Unusual Symptom of Maxillary Sinus Disease."**

BY G. J. DENNIS, M. D.

DISCUSSION.

DR. ELMER L. KENYON said that he is a subject of sinus disease himself, and feels that one should realize in considering cough in connection with maxillary sinus disease that cough is a symptom of any sinus disease.

One of the first things the experienced laryngologist considers in an acute case of sinus disease is whether or not secretion entering the nasopharynx is setting up cough. The maxillary is not the only sinus to produce symptoms of that kind and one should be very careful not to so consider it.

Referring to the inception of what appeared to be an infection in himself, he spoke of a persistent cough beginning at night upon going to bed. In the daytime he was practically comfortable and free from cough. He began to realize that he probably had a sinus disease. There had been no secretion coming out of the nose and he had no realization of a secretion going into the throat.

The diagnosis was infection of the posterior ethmoidal region on the left side. Later he had symptoms of asthma—his only experience.

The point made by him was that cough due to the irritation of the nasal pharynx from secretion passing into it, is a symptom of any sinus disease and is not at all peculiar to any one sinus.

DR. CHARLES H. LONG said that he had a maxillary sinusitis in which the cough was the predominating symptom. There was a general toxic condition and an irritation from the secretion passing down the throat. The voice was also affected, being lost almost completely when talking at any length.

He referred to the case of a young lady who had been the subject of a dry cough for several years, whose condition had been diagnosed as tuberculosis. She had been sent south for her health and had been treated by many specialists. Upon removal of the tonsils the cough was completely relieved, she

\*See page 1356.

regained her normal health and was cured of her "tuberculosis."

He emphasized the fact that other conditions cause cough as well as disease of the maxillary sinus. In the cases he has seen the cough is usually due to the secretion passing down the nasopharynx and causing irritation.

DR. FRANK J. NOVAK felt that the particularly significant observation in this paper of Dr. Dennis was that cough is an unusual symptom of sinus disease.

He believes that there is some fundamental basis for the cough shown in sinus disease, there is some reason for the hyperexcitability of this particular reflex, and that a study of the blood chemistry of these conditions might reveal the nature of the underlying condition.

DR. ALFRED LEWY thought the one case with the persistent spasmodic cough with profuse expectoration, suggested possible bronchiectasis, as that may be associated with sinus disease, and said that it would be interesting to know if Dr. Dennis had had at least some of these cases, that would suggest that condition, carefully gone over to eliminate that possibility.

He thought it would also be interesting to know whether or not in any case of bronchiectasis with sinus disease the cough would be minimized by the treatment of the sinus.

DR. CHARLES F. YERGER said that especially in maxillary sinus infections in tuberculous individuals, the cough may be due to the sinusitis rather than to the lung condition.

Knowing how frequently cough is present in tuberculous conditions of the lung, he referred to a certain group of cases in which we may have these two conditions present in the same individual. He has been able to relieve the cough in some of these cases of pulmonary tuberculosis with maxillary sinusitis where the sinus condition has cleared up and the lung condition also cleared up remarkably as a result of the cure of the sinusitis.

Every case of pulmonary tuberculosis should be examined with the idea that it may also have a sinus infection and if present the sinus should be treated. In many cases one may effect a cure of the symptoms which are present in sinus

disease and thus aid materially in the cure of pulmonary tuberculosis.

DR. HARRY KAHN said that while Dr. Dennis mentioned cough as a symptom of maxillary sinus disease, he believed he had not intended to convey the impression that cough does not also occur as a symptom of many other disorders. He thought Dr. Dennis should be given credit for bringing forward this rather unusual symptom of closed maxillary sinus disease.

DR. EDWARD P. NORCROSS had never had his attention called particularly to cough produced by maxillary sinus disease, but spoke of having produced a cough by forcing some air into a closed maxillary sinus. The cough lasted half an hour, with marked dyspnea. He could demonstrate no secretion that came out of the sinus at that time.

DR. HARRY L. POLLOCK thought Dr. Dennis wanted to demonstrate that this is a mere reflex cough from the sinus itself and not from other parts of the nose.

He referred to a paper given a year ago on complications of the antrum. Reference was made at that time to a Norwegian who had done a lot of work along this line. It was said that if a tracheotomy had been done previous to this antrum irritation and the trachea closed off entirely, that is, the pharynx closed off entirely, none of these symptoms developed. This man irritated the antrum in some animals, and a great many of the cases terminated fatally. On those animals that survived he did a tracheotomy and then did these experiments and found no results, trying to disprove it was not just the irritation of the antrum but the trickling of the solution down into the larynx that produced the trouble.

The irritation, Dr. Pollock felt, was not due to the irritation of the antrum itself but rather to the trickling of the solution, or water, or the secretion from the antrum down into the larynx. Sometimes very little is enough to set up the cough.

DR. G. J. DENNIS (closing) emphasized the fact that in the four cases there were no symptoms whatever in the nose; that there was no greater enlargement of the turbinates than one would find in the average nose and that by simply rubbing

a piece of cotton over the middle turbinate one can easily produce a cough.

He felt that the title of his paper might have been misleading and that it should have been "Cough as an Isolated Symptom of Maxillary Sinus Disease."

He had found nothing upon looking up the literature except one case and that was hardly in point, because there was secretion issuing from the sinus into the nose. The other cases reported also had secretion in the nose, but cough was the predominating symptom. He found a paper on bronchiectasis written by a doctor at Colorado Springs in 1921, "The Accessory Sinus as an Etiological Factor in Bronchiectasis."

He spoke of a thesis in which reference is made to twenty cases with rather disastrous results due to simple puncture of the antrum, four of the patients dying. In each of those cases there was interference with respiration together with cessation of the heart action.

Dr. Dennis found that much had been written about the reflex irritation of the nose itself, the writers carrying their conclusions to the point that the sphenopalatine ganglion is the center from which all these reflexes radiate. In studying the anatomy he found that the nerves which supply the sinuses do not enter the sphenopalatine ganglion but enter the ophthalmic division of the trigeminus. He thinks it would be of interest to determine whether those impulses are carried back to the ganglion. Such reflexes as we have must pass directly through the semilunar ganglion and from there apparently to the medulla oblongata, and the close relationship between the trigeminus and the vagus seems to make it an easy matter for reflexes to be conveyed to the vagus.

**\*Paper: "Left Temporal Lobe Abscess, With a Report of Two Cases."**

BY C. F. YERGER, M. D.

DISCUSSION.

DR. GEORGE W. BOOT referred to 73 cases of brain abscess reported by him four years ago. He has since added 12 cases; 9 from the County Hospital and 3 from private prac-

\*See age 1364.

tice. As a result of a study of these 85 case histories, he was in hearty agreement with everything said by the essayist.

He emphasized the fact that otitic brain abscess usually occurs as a result of extension from the ear, the brain abscess usually being found in the vicinity of the tympanum. If the extension is upward there is a temporal lobe abscess; if backward, a cerebellar abscess; in either case, within one inch of the original source of infection. Brain abscesses always occur in certain definite locations, very close to the original source of infection. There is an infection in the tympanic cavity, but one cannot tell how much the fever is due to that and how much is the result of infection in the brain itself.

The auditory center for the memory of names, first located by Charles K. Mills, of the University of Pennsylvania, some twenty-five or thirty years ago, is the center most commonly involved in a left temporal lobe abscess. The patient does not remember the names of objects, although he does know their use. It has not been decided positively that left-handed individuals have the word center in the right temporal lobe. Wernicke's hemiopic phenomenon is a very important symptom in some cases of temporal lobe abscess. This is obtained by throwing light into the pupil from one side or the other and seeing whether the ordinary pupillary response is present. If it is present in one side and not in the other, it is quite a definite indication of the location of the lesion.

In a case of suspected brain abscess he considers a slow pulse a very important symptom and a very definite indication for operation, if accompanied by other symptoms of brain abscess. It may be only relatively slow, as a temperature of 105° F., with a pulse of 70.

Lumbar puncture, when properly done, should not be dangerous and may give great information. In exceptional cases bacteria may be present. *Fungus cerebri* is not due to the size of the opening but to the infection of the brain substance itself, or cerebritis.

He thought the essayist did not sufficiently emphasize the importance of being careful not unnecessarily to traumatize the brain. He agreed that it is unwise to use gauze, as putting it in and removing it causes additional trauma to the brain, and thinks a rubber tube is very much to be preferred.



DR. ALBERT B. YUDELSON felt that a neurologic discussion of the subject would require a classification of septic lesions.

The symptomatology as given is of common occurrence—pain, temperature, vertigo and vomiting. The eye findings come somewhat later. If the process goes on without interference, intracranial pressure, naturally, produces a degree of optic atrophy. Auditory aphasia or word deafness comes later as the result of a blocking between the receiving station of words and the interpretation of their meaning. The auditory mechanism is not disturbed but the comprehension of the meaning of words is disturbed. That is a development rather than an early symptom.

The differential diagnosis between the localization of a brain abscess in the temporal lobe and the lesion in the internal capsule must be brought about by an examination of the sensory findings. There will be no anesthesia or analgesia on the opposite side in the case of a temporal lobe lesion. In the case of a capsular lesion there will be sensory disturbance because the fibers come in such close connection in the capsule that sensory fibers are caught in the septic or pathologic area.

He believed the importance of early diagnosis must be emphasized. An early recognition of the involvement in the case counts for everything.

The pathologic condition occurs by extension, following along lines of least resistance. When the symptoms of pain, vertigo, or percussion, indicate cerebellar extension; or if frontal headaches with optic findings indicate anterior extension, these points aid the surgeon in localizing the lesion and he should operate immediately. Free drainage is most helpful toward recovery.

DR. JOSEPH C. BECK considered the Society very fortunate in having four men on the attending staff of one of the best institutions in the country, where brain abscess is of sufficient frequency for a study of the question, and in having conclusions of the work presented by Dr. Yerger.

The question of brain abscess must be considered where it is secondary to operations due to exposure of the dura and operative trauma of the dura where there is no abscess at the time of operation.

The pain referable to brain abscess is not always at the location of the abscess; there may be symptoms that are well recognized as *contra coup*. The abscess may be localized in the temporosphenoidal lobe of one side and produce its symptoms on the opposite side.

Dr. Beck asked if the essayist had encountered paralysis in cases of sphenoid abscess, and cited two such cases in which the abducens, from its long course located quite close to the bone surface, produced paralysis. He also requested that Dr. Yudelsohn be asked to touch upon the psychic symptoms, as he had often seen manifested in the way of foolish remarks in these cases.

He took issue with Dr. Boot, who said that it does not make any difference how big a hole is made and thought that the theory would not hold. If a large section of the skull is removed and the dura exposed the pressure is still greater than normal and the danger of herniation is increased, although frequently the opening is not made large enough to serve the desired purpose. He thought it inadvisable to open any unprotected areas above in the temporal region in an effort to reach an abscess when there is such an excellent way of reaching it through the tegmen. That route prevents making too large a hole, and there is not as much danger of herniation as when opening in the temporal area.

DR. SAMUEL SALINGER thought the emphasis laid upon the symptoms of aphasia would lead one to believe that the majority of the abscesses occur on the left side, whereas postmortem records have shown that an average of 60 to 65 per cent of brain abscesses occur on the right side. If a great deal of stress is laid on that one symptom, a number of abscesses may be overlooked in which this symptom is absent.

Neither should too great emphasis be laid on the location of the abscess immediately contiguous to the abscessed area of bone, experience having shown there are quite a number of cases in which the abscess is located at a distant point; in fact, as far distant as the opposite hemisphere. One cannot always count on the adjacent brain tissue being infected. Many times upon opening the dura the brain tissue is found to be perfectly normal and the abscess located at a considerable distance. There is also the abscess which occurs on the sur-

face of the cortex, the so-called intrapiaarachnoid. He has seen this occur over the frontal lobe secondary to an otitis media, certainly far enough removed. These various types should be taken into consideration, each case being considered on its own merits. One should not cleave to one classic example because exceptions are very frequent.

DR. HARRY BOYD-SNEE referred to the necropsy findings in eight cases he has had under his observation. It was found they were not dealing with a single subcortical suppurative focus or a single brain abscess. In three of the cases they found multiple subcortical brain abscess.

The cases that came to his notice developed connection with an acute disease process originating in the ear, the brain abscess developing at periods varying from six weeks to six months after the onset of those acute processes, or after mastoid operation had been done. All of these cases terminated fatally and were checked by necropsy findings.

He asked the essayist whether in the case reported of leptomeningitis developing two months after operation, there was an autopsy to determine whether or not he had dealt with a single or multiple brain abscess proposition.

DR. G. J. DENNIS referred to a case under his observation at the present time in a child, aged eight years, with a suppurative right ear which healed with closure of the drum a few days before. About two weeks ago the child had chills lasting an hour; a temperature running to 105° F. and absence of pupillary reflexes and corneal reflexes lasting an hour or so. Then the child was apparently in good condition for four or five days, when the symptoms became more frequent and lasted very much longer.

A diagnosis was made of either subdural abscess or sinus thrombus of the right side, and a mastoid operation was done that night. The mastoid was of ivory firmness with an antrum filled with granulation tissue but no pus. The lateral sinus was uncovered for nearly two inches and examining by puncture, no thrombosis was found. Separation for about four inches in every direction revealed no meningitis or abscess. There were no focal symptoms in the beginning.

The literature on the subject shows that very few of these right-sided brain abscesses show any focal symptoms. Brain

abscesses usually have a continuous temperature, not over 102° F., but sinus thrombosis usually has an irregular temperature, running up to 105° F.

DR. CHARLES F. YERGER (closing) in answer to Dr. Boot, emphasized the point that there may be no fever or that there may be a subnormal temperature. He has seen brain abscess cases of both types, without any fever and with a subnormal temperature.

Wernicke's hemiopic pupillary sign is a very difficult test to obtain and it has always been difficult for him to be sure of this test. It is superfluous when the visual fields can be obtained.

Replying to Dr. Beck, he thinks all are agreed that hernia of the brain is produced by a large opening in the dura. The dura being a dense membrane holding the brain substance back, if a large incision is made into the dura that allows the brain to herniate. So he believes that the size of the opening in the dura is more important than the size of the opening in the bone.

Referring to Dr. Beck's point about brain abscess secondary to operation, he spoke of a case of brain abscess probably produced at operation in which the patient recovered. However, there was a sympathetic meningitis and operation had been necessary for removal of the focus of infection external to the dura. Not being satisfied with the negative findings, he went through the dura.

He emphasized that one must be very careful about going through the dura. The brain tissue can be easily infected by going through an infected region like the mastoid, opening the dura and probing the brain.

He has not had a case of abducens nerve paralysis in his limited experience with brain abscesses. He has had a case of amnesic aphasia but omitted that subject from the paper. The individual did not know the town in which she was born or her mother's name.

Dr. Yerger agreed with Dr. Salinger about abscesses occurring most frequently on the right side; also about abscesses being distant from the dura and bone involved. His paper was intended as a discussion of the left temporal lobe abscess with emphasis on the aphasic symptoms.

Replying to Dr. Boyd-Snee, Dr. Yerger said that multiple abscess usually is not a direct extension but occurs more frequently through the circulation. He called attention to one of Dr. Beck's cases of multiple abscesses broadcast, he believed, by the circulatory system.

In the case cited in his paper autopsy was performed and a single abscess was found. This patient died of septic meningitis two months after the operation.

**\*Paper: "Intranasal Syphilis."**

By C. H. CHRISTOPH, M. D.

DISCUSSION.

DR. EDWARD P. NORCROSS called attention to the importance of diseases of the upper respiratory tract and ear in connection with other general diseases.

Cases are presented to the laryngologist for some local condition, the correct diagnosis of which reveals some condition the patient had not even suspected. A diagnosis of laryngeal tuberculosis frequently gives the patient the first intimation that he has a pulmonary tuberculosis. By the use of tuning forks one often is able to detect that the patient has syphilis. Intranasal syphilis is frequently the first diagnosis made of syphilis. He was somewhat surprised to have the essayist speak of the rarity of primary lesions of the nose. In the last three or four years Dr. Norcross has had two cases of primary syphilis of the septum. In the first case a young woman complained of a rather sudden obstruction to her nasal breathing and some headache. Examination of the nose showed an infiltrated mass on one side of the septum. The character of the lesion and the course of the disease did not resemble an ordinary rhinitis, and while being treated she developed a secondary rash which covered the whole body.

A little more than a year ago a physician who had trouble breathing through his left nostril came to him for treatment. Examination revealed an infiltrated mass on the left side of the septum, some bleeding and a secondary glandular involvement of the neck. The patient was referred to Dr. Zeisler,

\*See page 1379.

and smears of the lesion showed the spirocheta pallida. Under antisyphilitic treatment he recovered.

Dr. Zeisler said he had had only one other case and that was a physician, and that his father, in his extensive practice, had only one case of a primary lesion of the septum.

The essayist mentioned postmortem records that  $2\frac{1}{2}$  per cent to 3 per cent of syphilitic cases have involvement of the nose. The most frequent lesion that the physician sees is the tertiary lesion, occurring any time from a year to a matter of twenty, thirty or forty years after the primary infection. The unfortunate thing is that the tertiary lesion produces so few symptoms that the patient does not present himself until there is a great deal of destruction of the nose. The man who thinks he has syphilis comes in with every abrasion of the nose to see if it is syphilitic.

The first symptom that may bring the actual syphilitic to the physician is the fact that fluids regurgitate through the nose, or possibly the stench from the dead bone is so great that that is his complaint. One never forgets the smell of syphilitic necrotic bone.

The lack of symptoms is one of the characteristic findings in nasal syphilis, but the results of untreated cases are so disastrous that one should be very careful in making diagnoses. Not only are such cases very distressing to the patient but he goes about with his diagnosis where everyone can read it.

A favorite site for syphilis is at the junction of the hard and soft palate, and due to the rapid destruction of the bone here the nose is very soon involved.

Secondary lesions are not very commonly seen in the nose. The white patches may occur in the mouth but are not so commonly seen in the nose. It is said that when a secondary lesion does appear in the nose, if secondary to a primary lesion in the same location, its destruction may be almost as great as the tertiary lesion. The lesion is more often confined to the destruction of bone rather than to cartilage alone. One may see the whole septum destroyed with no external deformity, but the deformity seems to appear when the nasal bones themselves are attacked.

DR. ERWIN P. ZEISLER said they had had three cases of chancre of the nasal septum in their office. Some years ago his father had a patient referred by Dr. Campbell with a primary lesion of the septum associated with enlarged glands in the cervical region and a secondary syphiloderma.

Of the two cases with which he had had experience one was that mentioned by Dr. Norcross, a physician with an ulcer of the septum with glandular enlargement in the submaxillary region. He examined for spirochetes and found them in the dark field. The Wassermann reaction also was positive.

The third case was that of a physician in the same specialty as members of the Society. He had a generalized nodular eruption which had not been diagnosed on account of the apparent absence of a primary lesion. The finding of a hard mass of glands in one side of the neck led to the discovery of an ulceration of the septum which was the primary lesion. The diagnosis of a chancre of the nasal septum should be made clinically. In his experience the lesion is not fungoid, but an ulcer which does not heal. The presence of a hard pocket of glands in the submaxillary region is of great importance in the diagnosis. It should not be difficult to find the spirochetes in intranasal chancre, provided local antiseptics have not been used. In the throat there may be some difficulty in differentiating the *spirocheta pallida* from other spirochetes. Only a few weeks ago they had a patient at the County Hospital on whom he made a clinical diagnosis of chancre of the tonsil in spite of repeated negative spirochete reports. Yet after a few weeks the nodular eruption appeared and the Wassermann reaction became positive.

In regard to differentiation of intranasal lupus from late syphilis it is important to remember that lupus destroys skin, subcutaneous tissue and cartilage, but leaves the bone intact. On the other hand, syphilis attacks the bony structures with great frequency. Furthermore, there will always be found evidence of lupus in the skin around the nose. In his experience lupus of the septum of the nose was rare in this country.

In the examination of old luetic patients it is very important to examine the septum of the nose in a routine fashion. One may find a small perforation in the septum or in the hard

palate which is practically pathognomonic. The Wassermann reaction in many of these cases may be negative, particularly in those that have had treatment.

DR. GEORGE W. BOOT said that he had had two cases of gumma of the frontal sinus. He was fortunate to get one case before there was any breaking down of the tissue. The other patient had a suppurative sinus on the left side with an excessively foul discharge. He operated and removed a portion of the skull, 5 x 7.5 cm. The man had no brain symptoms, although the dura had been bathed in pus for weeks.

A case of tertiary syphilis of the nose had destroyed the entire interior of the nose as well as the exterior. This case was very instructive with reference to the action of the soft palate. Instead of the velum closing the nasopharynx like a valve it closes like a sphincter.

With reference to syphilis, a woman living in one of the exclusive suburbs of Chicago came to the doctor for treatment of a cold in the nose. She was given some little yellow pills by the doctor with happy results. Soon she had all the women in the neighborhood taking these little yellow pills for colds.

With reference to Hutchinson's teeth, he said he recently had a case where there had been a great deal of family discord partly due to a diagnosis of Hutchinson's teeth. The wife had been attended in her first confinement by a doctor who used his instruments rather clumsily, and he claimed that her puerperal infection was because her husband had syphilis. The husband had repeated Wassermann tests made and they were always negative. He never had any symptoms of syphilis. When the child was some five years old there was a diagnosis of Hutchinson's teeth due to hereditary syphilis. Hutchinson's teeth are the second teeth, not the first, and she had not cut her second teeth at the time the diagnosis was made. The first teeth are often poorly formed if the child was poorly nourished in infancy or had any of the exanthemata but such teeth are not Hutchinson's teeth, and a diagnosis of hereditary syphilis cannot be made from the first teeth.

DR. EDWIN MCGINNIS brought out the point that one of the surgeons of his acquaintance thought it peculiar that sequestra form in the nose from tertiary syphilis but very



rarely anywhere else in the osseous system. This may be due to secondary infections that do not occur anywhere else.

Another point brought out was in regard to an operation he did for an older laryngologist on a syphilitic septum. There was quite a perforation after the operation. There had not been a Wassermann test because Dr. McGinnis was just called over to operate. During the past year this patient reported that he had "something loose" in his nose. On examination it was found that the middle turbinate and most of the ethmoidal labyrinth on one side had loosened and were lying on the floor of the nose. The Wassermann reaction was 4+, and the reason for the perforation of the septum following the operation became apparent seven or eight years after the operation.

DR. CARL H. CHRISTOPH (closing) said that the old classic description of intranasal syphilis, which describes the destruction of the bone, is really not applicable to all cases. Syphilis is not suspected in many cases in which there is no bone involvement but which are definitely syphilitic, such as old chronic sinuses and other obscure nasal conditions which do not get well by the ordinary methods of treatment but which clear up when antisyphilitic measures are instituted.